

=> d his full

(FILE 'HOME' ENTERED AT 12:27:40 ON 17 JUL 2006)

FILE 'REGISTRY' ENTERED AT 12:27:59 ON 17 JUL 2006

E FERRIOX/CN

L1 1 SEA ABB=ON PLU=ON FERRIOXAMINE/CN
D SCA
L2 1 SEA ABB=ON PLU=ON FERRIOXAMINE B/CN
D SCA
L3 1 SEA ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4 1 SEA ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5 1 SEA ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6 2 SEA ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7 6 SEA ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5 OR L6)
E TRIHYDROXAMI/CN
E HYDROXAMI/CN
E CP94/CN
E CP 94/CN

L8 1 SEA ABB=ON PLU=ON CP 94/CN
D SCA
E EDTA/CN

L9 1 SEA ABB=ON PLU=ON EDTA/CN
D SCA

L10 1 SEA ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN
D SCA

L*** DEL 1 S L9-L10
E DEFEROX/CN

L11 1 SEA ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN

L12 0 SEA ABB=ON PLU=ON L11 AND L7

L13 1 SEA ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN OR "DEFEROXAMIN
E METHANESULFONATE"/CN)

L14 0 SEA ABB=ON PLU=ON L13 AND L7
E DESFERAL/CN

L15 1 SEA ABB=ON PLU=ON DESFERAL/CN
D SCA

L16 1 SEA ABB=ON PLU=ON DESFERAL M?/CN
E APOFERRITIN/CN

L17 1 SEA ABB=ON PLU=ON APOFERRITIN?/CN
E CDTA/CN

L18 1 SEA ABB=ON PLU=ON CDTA/CN
D SCA
E DTPA/CN

L19 1 SEA ABB=ON PLU=ON DTPA/CN
E PENICILLAMIN/CN

L20 1 SEA ABB=ON PLU=ON PENICILLAMINE/CN
D SCA
E BATHOCUPROIN/CN
E BATHOCUPPROIN/CN

L21 6 SEA ABB=ON PLU=ON BATHOCUP?/CN
E DIETHYLENETRIAMINE/CN

L22 4 SEA ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETIC?/CN

L23 23 SEA ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR
L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR
L17 OR L18 OR L19 OR L20 OR L21 OR L22)
D COST

FILE 'STNGUIDE' ENTERED AT 12:42:46 ON 17 JUL 2006

FILE 'STNGUIDE' ENTERED AT 12:53:16 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 12:54:03 ON 17 JUL 2006
E US2003-617943/APPS

L24 1 SEA ABB=ON PLU=ON US2003-617943/APPS
D SCA
D IALL

FILE 'STNGUIDE' ENTERED AT 12:54:39 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 12:57:00 ON 17 JUL 2006
SEL RN

FILE 'REGISTRY' ENTERED AT 12:57:13 ON 17 JUL 2006

L25 36 SEA ABB=ON PLU=ON (138-14-7/BI OR 70-51-9/BI OR 115900-75-9/B
I OR 117-39-5/BI OR 13291-61-7/BI OR 146426-40-6/BI OR
14836-73-8/BI OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI
OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR
482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR
50-78-2/BI OR 52-67-5/BI OR 520-26-3/BI OR 520-27-4/BI OR
520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR 525-82-6/BI OR
577-85-5/BI OR 60-00-4/BI OR 67-43-6/BI OR 73348-75-1/BI OR
7439-89-6/BI OR 7440-50-8/BI OR 7447-39-4/BI OR 75-91-2/BI OR
7758-94-3/BI OR 989-51-5/BI)

L26 8 SEA ABB=ON PLU=ON L25 AND L23

FILE 'HCAPLUS' ENTERED AT 12:57:43 ON 17 JUL 2006

L27 37763 SEA ABB=ON PLU=ON L26

L28 1 SEA ABB=ON PLU=ON L24 AND L27
D SCA

FILE 'REGISTRY' ENTERED AT 12:58:24 ON 17 JUL 2006

L29 1 SEA ABB=ON PLU=ON 70-51-9

FILE 'HCAPLUS' ENTERED AT 12:58:37 ON 17 JUL 2006

L30 2619 SEA ABB=ON PLU=ON L29

FILE 'REGISTRY' ENTERED AT 12:58:52 ON 17 JUL 2006

L31 24 SEA ABB=ON PLU=ON L23 OR L29

FILE 'HCAPLUS' ENTERED AT 13:32:52 ON 17 JUL 2006

L32 40989 SEA ABB=ON PLU=ON L31

L33 1 SEA ABB=ON PLU=ON L24 AND L32
D SCA

FILE 'REGISTRY' ENTERED AT 13:33:45 ON 17 JUL 2006

L34 27 SEA ABB=ON PLU=ON L25 NOT L31

FILE 'HCAPLUS' ENTERED AT 13:34:06 ON 17 JUL 2006

L35 16008 SEA ABB=ON PLU=ON L34 (L) THU/RL

L36 1 SEA ABB=ON PLU=ON L24 AND L35
D SCA
SEL HIT RN

FILE 'REGISTRY' ENTERED AT 13:35:00 ON 17 JUL 2006

L37 22 SEA ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/BI OR 153-18-4/
BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/B
I OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI
OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR 520-26-3/BI OR
520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR
525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)

L38 1 SEA ABB=ON PLU=ON 50-78-2
 L39 21 SEA ABB=ON PLU=ON L37 NOT L38

FILE 'HCAPLUS' ENTERED AT 13:35:37 ON 17 JUL 2006
 L40 34358 SEA ABB=ON PLU=ON L39
 L41 1 SEA ABB=ON PLU=ON L40 AND L24
 D SCA

FILE 'STNGUIDE' ENTERED AT 13:36:14 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 13:36:39 ON 17 JUL 2006
 E ATAXIA TELANGIECTASIA+ALL/CT
 E E2+ALL

L42 1665 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/OBI
 L43 2356 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BI
 L44 0 SEA ABB=ON PLU=ON LOUIS BAR/OBI
 L45 8 SEA ABB=ON PLU=ON LOUIS BAR/BI
 L46 8 SEA ABB=ON PLU=ON LOUIS-BAR/BI
 L47 0 SEA ABB=ON PLU=ON CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
 L48 0 SEA ABB=ON PLU=ON CEREBELLO OCULOT?/BI

FILE 'STNGUIDE' ENTERED AT 13:39:59 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 13:40:24 ON 17 JUL 2006
 L49 2363 SEA ABB=ON PLU=ON (ATAXIA (2A) TELANGIECT?)/BI
 E CHELATING AGENT+ALL/CT
 E CHELATING AGENTS+ALL/CT
 L50 15362 SEA ABB=ON PLU=ON CHELATING AGENTS+OLD,NT/CT
 L51 40989 SEA ABB=ON PLU=ON L31
 L52 2369 SEA ABB=ON PLU=ON (L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR
 L48 OR L49)
 L53 52491 SEA ABB=ON PLU=ON (L50 OR L51)
 L54 7 SEA ABB=ON PLU=ON L52 AND L53
 D SCA
 L55 34358 SEA ABB=ON PLU=ON L39
 L56 4 SEA ABB=ON PLU=ON L54 AND L55
 L57 144038 SEA ABB=ON PLU=ON ANTIOXID?/BI
 E FLAVONOIDS+ALL/CT
 E E2+ALL/CT
 L58 57074 SEA ABB=ON PLU=ON FLAVONOIDS+NT,OLD,UF/CT
 L59 34491 SEA ABB=ON PLU=ON FLAVONOIDS?/BI
 L60 4 SEA ABB=ON PLU=ON L54 AND (L57 OR L58 OR L59)
 D SCA
 L61 11651 SEA ABB=ON PLU=ON ?HYDROXAMIC ACID?/BI
 E FERRITINS+ALL/CT
 L62 9439 SEA ABB=ON PLU=ON FERRITINS+OLD,UF/CT
 L63 17 SEA ABB=ON PLU=ON L52 AND (L61 OR L62)
 L64 14 SEA ABB=ON PLU=ON L63 NOT L54
 L65 374 SEA ABB=ON PLU=ON FERRITINS/CT (L) (THU OR BAC OR DMA OR PAC
 OR PKT)/RL
 L66 5 SEA ABB=ON PLU=ON L65 AND L52
 L67 3 SEA ABB=ON PLU=ON L66 NOT L54
 D SCA
 D SCA TI
 E HYDROXAMIC ACIDS+ALL/CT
 L68 15322 SEA ABB=ON PLU=ON HYDROXAMIC ACIDS+NT/CT
 L69 29 SEA ABB=ON PLU=ON L68 AND L52
 L70 132130 SEA ABB=ON PLU=ON CHELAT?/BI
 L71 3 SEA ABB=ON PLU=ON L69 AND L70
 D SCA

L72 6 SEA ABB=ON PLU=ON L70 AND L52
 L73 1 SEA ABB=ON PLU=ON L72 NOT L54
 D SCA
 L74 29887 SEA ABB=ON PLU=ON WANG S?/AU
 L75 49 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L76 12 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L77 5 SEA ABB=ON PLU=ON L74 AND (L75 OR L76)
 L78 12 SEA ABB=ON PLU=ON L52 AND (L74 OR L75 OR L76)

FILE 'MEDLINE' ENTERED AT 14:04:08 ON 17 JUL 2006

D COST

L79 9295 SEA ABB=ON PLU=ON WANG S?/AU
 L80 81 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L81 10 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L82 3 SEA ABB=ON PLU=ON L79 AND (L80 OR L81)
 E ATAXIA TELANGIECTASIA+ALL/CT
 L83 2457 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
 L*** DEL 3951 S ATAXIA TELANGIECTAS?
 D TRIAL 1-3
 L84 3932 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L85 3935 SEA ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
 L86 3935 SEA ABB=ON PLU=ON (L83 OR L84 OR L85)
 L87 13 SEA ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
 E CHELATING AGENTS+ALL/CT
 L88 13204 SEA ABB=ON PLU=ON CHELATING AGENTS/CT
 L89 92986 SEA ABB=ON PLU=ON CHELATING AGENTS+NT/CT
 L90 3231 SEA ABB=ON PLU=ON IRON CHELATING AGENTS/CT
 L91 19528 SEA ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
 E IRON CHELATING AGENTS+ALL/CT
 L*** DEL 0 S SIDEPHORES/CT
 L*** DEL 0 S SIDEPHORES+NT/CT
 L92 1267 SEA ABB=ON PLU=ON SIDEROPHORES/CT
 L93 6055 SEA ABB=ON PLU=ON SIDEROPHORES+NT/CT

FILE 'REGISTRY' ENTERED AT 14:12:03 ON 17 JUL 2006

SET SMARTSELECT ON

L94 SEL PLU=ON L31 1- CHEM : 255 TERMS
 SET SMARTSELECT OFF

FILE 'MEDLINE' ENTERED AT 14:12:07 ON 17 JUL 2006

L95 68933 SEA ABB=ON PLU=ON L94
 L96 7 SEA ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90 OR L91 OR L92
 OR L93) OR L95)
 D TRIAL 1-7
 L97 37091 SEA ABB=ON PLU=ON CHELAT?
 L98 5 SEA ABB=ON PLU=ON L86 AND L97
 L99 0 SEA ABB=ON PLU=ON L98 NOT L96
 L100 61053 SEA ABB=ON PLU=ON ANTIOXID?
 L101 18939 SEA ABB=ON PLU=ON FLAV!NOID?/BI
 L102 32882 SEA ABB=ON PLU=ON FLAVONOIDS+NT/CT
 L*** DEL 0 S FLAVANOIDS+NT/CT
 L103 QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
 L104 3 SEA ABB=ON PLU=ON L96 AND ((L100 OR L101 OR L102))
 D TRIAL 1-3
 L105 3 SEA ABB=ON PLU=ON L96 AND ((L100 OR L101 OR L102 OR L103))
 D TRIAL 1-3

FILE 'REGISTRY' ENTERED AT 14:18:45 ON 17 JUL 2006

SET SMARTSELECT ON

L106 SEL PLU=ON L39 1- CHEM : 344 TERMS

SET SMARTSELECT OFF

FILE 'MEDLINE' ENTERED AT 14:18:48 ON 17 JUL 2006

L107 19788 SEA ABB=ON PLU=ON L106
 L108 2 SEA ABB=ON PLU=ON L107 AND L96
 D TRIAL 1-2
 L109 QUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESFERROXAMIN?
 IN? OR DEFERRIOXAMIN?
 L110 QUE ABB=ON PLU=ON EDETIC ACID/CT
 L111 QUE ABB=ON PLU=ON CP94
 L112 QUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
 L113 QUE ABB=ON PLU=ON APOFERRITIN/CT
 L114 QUE ABB=ON PLU=ON CDTA
 L115 QUE ABB=ON PLU=ON DTPA OR PENTATRIC ACID
 L116 QUE ABB=ON PLU=ON PENICILLAMINE
 L117 QUE ABB=ON PLU=ON BATHOCUPROINE
 L118 QUE ABB=ON PLU=ON BATHOCUPROIN
 L119 6 SEA ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111 OR L112 OR
 L113 OR L114 OR L115 OR L116 OR L117 OR L118)
 D TRIAL 1-6
 L120 1 SEA ABB=ON PLU=ON L119 AND L107
 D TRIAL
 L121 13 SEA ABB=ON PLU=ON L82 OR L87
 L122 10 SEA ABB=ON PLU=ON L96 OR L98 OR L105 OR L108 OR L119 OR L120
 L123 7 SEA ABB=ON PLU=ON L122 NOT L121

FILE 'EMBASE' ENTERED AT 14:31:14 ON 17 JUL 2006

L124 7035 SEA ABB=ON PLU=ON WANG S?/AU
 L125 5 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L126 27 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L127 4 SEA ABB=ON PLU=ON L124 AND (L125 OR L126)
 E ATAXIA TELANGIECTASIA/CT
 E ATAXIA TELANGIECTASIA+ALL/CT
 E ATAXIA TELANGIECTASIA+UF/CT
 L128 2332 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
 L129 3044 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L130 62 SEA ABB=ON PLU=ON LOUIS BAR
 L131 2 SEA ABB=ON PLU=ON ATAXIA TELANGIECTATICA
 L132 0 SEA ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCUTANEA
 L133 0 SEA ABB=ON PLU=ON TELANGIECTASIA CEREBELLO OCULOCUTANEA
 L134 3053 SEA ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131 OR L132 OR
 L133)
 L135 11 SEA ABB=ON PLU=ON (L124 OR L125 OR L126) AND L134
 E CHELATING AGENT+ALL/CT
 L136 98621 SEA ABB=ON PLU=ON CHELATING AGENT+NT/CT

FILE 'REGISTRY' ENTERED AT 14:35:53 ON 17 JUL 2006

SET SMARTSELECT ON

L137 SEL PLU=ON L31 1- CHEM : 255 TERMS
 SET SMARTSELECT OFF

FILE 'EMBASE' ENTERED AT 14:35:55 ON 17 JUL 2006

L138 61283 SEA ABB=ON PLU=ON L137
 L139 14 SEA ABB=ON PLU=ON L134 AND (L136 OR L138)
 D TRIAL 1-14
 E FLAVONOIDS+ALL/CT
 L140 25033 SEA ABB=ON PLU=ON FLAVONOIDS+NT/CT
 E ANTIOXIDANT+ALL/CT
 L141 35447 SEA ABB=ON PLU=ON ANTIOXIDANT+NT/CT

L142 4 SEA ABB=ON PLU=ON L139 AND (L140 OR L141)
 D TRIAL 1-4

FILE 'REGISTRY' ENTERED AT 14:41:15 ON 17 JUL 2006
 SET SMARTSELECT ON
 L143 SEL PLU=ON L39 1- CHEM : 344 TERMS
 SET SMARTSELECT OFF

FILE 'EMBASE' ENTERED AT 14:41:16 ON 17 JUL 2006
 L144 24101 SEA ABB=ON PLU=ON L143
 L145 2 SEA ABB=ON PLU=ON L139 AND L144
 D TRIAL
 D TRIAL 2
 L146 14 SEA ABB=ON PLU=ON L139 OR L142 OR L145
 L147 11 SEA ABB=ON PLU=ON L127 OR L135
 L148 10 SEA ABB=ON PLU=ON L146 NOT L147
 D TRIAL 1-5
 L149 31221 SEA ABB=ON PLU=ON CHELAT?
 L150 4 SEA ABB=ON PLU=ON L149 AND L134
 D TRIAL
 D TRIAL 2-4

FILE 'BIOSIS' ENTERED AT 14:47:22 ON 17 JUL 2006
 L*** DEL 4 S WANS S?/AU
 L151 10521 SEA ABB=ON PLU=ON WANG S?/AU
 L152 52 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L153 7 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L154 6 SEA ABB=ON PLU=ON L151 AND (L152 OR L153)
 L155 3180 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L156 18 SEA ABB=ON PLU=ON (L151 OR L152 OR L153) AND L155
 E CHELATING AGENTS+ALL/CT
 E E3+ALL
 L157 38028 SEA ABB=ON PLU=ON CHELAT?

FILE 'REGISTRY' ENTERED AT 14:50:27 ON 17 JUL 2006
 SET SMARTSELECT ON
 L158 SEL PLU=ON L31 1- CHEM : 255 TERMS
 SET SMARTSELECT OFF

FILE 'BIOSIS' ENTERED AT 14:50:28 ON 17 JUL 2006
 L159 59051 SEA ABB=ON PLU=ON L158
 L160 3182 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
 L161 4049 SEA ABB=ON PLU=ON TRANSITION METAL?
 L162 160 SEA ABB=ON PLU=ON TRANSITION ELEM?
 L163 3396 SEA ABB=ON PLU=ON SIDEROPHOR?
 L164 42 SEA ABB=ON PLU=ON SIDEROCHROM?
 L165 72 SEA ABB=ON PLU=ON LOUIS BAR
 L166 3223 SEA ABB=ON PLU=ON L155 OR L160 OR L165
 L167 QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
 L168 7 SEA ABB=ON PLU=ON L166 AND L167
 D SCA
 L169 82137 SEA ABB=ON PLU=ON ANTIOXID? OR FLAV!NOID?

FILE 'REGISTRY' ENTERED AT 14:55:16 ON 17 JUL 2006
 SET SMARTSELECT ON
 L170 SEL PLU=ON L39 1- CHEM : 344 TERMS
 SET SMARTSELECT OFF

FILE 'BIOSIS' ENTERED AT 14:55:17 ON 17 JUL 2006
 L171 31206 SEA ABB=ON PLU=ON L170

L172 3 SEA ABB=ON PLU=ON L171 AND L168
 D SCA
 L173 0 SEA ABB=ON PLU=ON L168 AND (L161 OR L162)
 L174 18 SEA ABB=ON PLU=ON L154 OR L156
 L*** DEL 76 S L168 OR L172 OR L!73
 L175 7 SEA ABB=ON PLU=ON L168 OR L172 OR L173
 L176 4 SEA ABB=ON PLU=ON L174 AND L175
 D SCA

FILE 'EMBASE' ENTERED AT 14:58:18 ON 17 JUL 2006

L177 14 SEA ABB=ON PLU=ON L146 OR L150
 L178 4 SEA ABB=ON PLU=ON L147 AND L177

FILE 'MEDLINE' ENTERED AT 14:59:22 ON 17 JUL 2006

L179 3 SEA ABB=ON PLU=ON L121 AND L122

FILE 'HCAPLUS' ENTERED AT 15:00:01 ON 17 JUL 2006

L180 12 SEA ABB=ON PLU=ON (L77 OR L78)
 L181 7 SEA ABB=ON PLU=ON L54 OR L56 OR L71
 L182 4 SEA ABB=ON PLU=ON L180 AND L181

FILE 'STNGUIDE' ENTERED AT 15:00:33 ON 17 JUL 2006

FILE 'USPATFULL' ENTERED AT 15:01:27 ON 17 JUL 2006

L183 5870 SEA ABB=ON PLU=ON L31
 L184 2355 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L185 6 SEA ABB=ON PLU=ON L183 AND L184
 D SCA
 L*** DEL 0 S L31 (L) THU/RL
 D KWIC 1-6
 L186 1795 SEA ABB=ON PLU=ON L39
 L187 1 SEA ABB=ON PLU=ON L185 AND L186

FILE 'WPIX' ENTERED AT 15:05:21 ON 17 JUL 2006

L188 247 SEA ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
 L189 5402 SEA ABB=ON PLU=ON WANG S?/AU
 L190 0 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L191 3 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L192 1 SEA ABB=ON PLU=ON L189 AND (L190 OR L191)
 D SCA
 SEL DCN
 L193 0 SEA ABB=ON PLU=ON (RAAMBT/DCR OR RAGNQ8/DCR OR RA0DFA/DCR OR
 RA0EMC/DCR OR RA0JBK/DCR OR RA00TF/DCR OR RA0055/DCR OR
 RA021P/DCR OR RA0529/DCR OR RA1HHQ/DCR OR RA1XA5/DCR OR
 RA37W9/DCR OR R00064/DCR OR R00195/DCR OR R00268/DCR OR
 R00971/DCR OR R01179/DCR OR R01318/DCR OR R01319/DCR OR
 R03811/DCR OR R03812/DCR OR R03949/DCR OR R04870/DCR OR
 R06069/DCR OR R06174/DCR OR R06413/DCR OR R06747/DCR OR
 R07001/DCR OR R07027/DCR OR R08105/DCR OR R08504/DCR OR
 R09163/DCR OR R09222/DCR OR R09884/DCR OR R11605/DCR OR
 R19085/DCR OR R19452/DCR OR R20811/DCR OR R22037/DCR)
 L194 0 SEA ABB=ON PLU=ON (RAAMBT/DCRE OR RAGNQ8/DCRE OR RA0DFA/DCRE
 OR RA0EMC/DCRE OR RA0JBK/DCRE OR RA00TF/DCRE OR RA0055/DCRE OR
 RA021P/DCRE OR RA0529/DCRE OR RA1HHQ/DCRE OR RA1XA5/DCRE OR
 RA37W9/DCRE OR R00064/DCRE OR R00195/DCRE OR R00268/DCRE OR
 R00971/DCRE OR R01179/DCRE OR R01318/DCRE OR R01319/DCRE OR
 R03811/DCRE OR R03812/DCRE OR R03949/DCRE OR R04870/DCRE OR
 R06069/DCRE OR R06174/DCRE OR R06413/DCRE OR R06747/DCRE OR
 R07001/DCRE OR R07027/DCRE OR R08105/DCRE OR R08504/DCRE OR
 R09163/DCRE OR R09222/DCRE OR R09884/DCRE OR R11605/DCRE OR

R19085/DCRE OR R19452/DCRE OR R20811/DCRE OR R22037/DCRE)

FILE 'STNGUIDE' ENTERED AT 15:09:00 ON 17 JUL 2006

FILE 'WPIX' ENTERED AT 15:09:49 ON 17 JUL 2006

L195 5533 SEA ABB=ON PLU=ON (RAAMBT/DCN OR RAGNQ8/DCN OR RA0DFA/DCN OR
 RA0EMC/DCN OR RA0JBK/DCN OR RA00TF/DCN OR RA0055/DCN OR
 RA021P/DCN OR RA0529/DCN OR RA1HHQ/DCN OR RA1XA5/DCN OR
 RA37W9/DCN OR R00064/DCN OR R00195/DCN OR R00268/DCN OR
 R00971/DCN OR R01179/DCN OR R01318/DCN OR R01319/DCN OR
 R03811/DCN OR R03812/DCN OR R03949/DCN OR R04870/DCN OR
 R06069/DCN OR R06174/DCN OR R06413/DCN OR R06747/DCN OR
 R07001/DCN OR R07027/DCN OR R08105/DCN OR R08504/DCN OR
 R09163/DCN OR R09222/DCN OR R09884/DCN OR R11605/DCN OR
 R19085/DCN OR R19452/DCN OR R20811/DCN OR R22037/DCN)
 L196 0 SEA ABB=ON PLU=ON L188 AND (L189 OR L190 OR L191)
 L197 2 SEA ABB=ON PLU=ON L188 AND L195
 D SCA
 D HIT L197 1-2
 SEL HIT DCN
 L198 623 SEA ABB=ON PLU=ON (R06069-/DCN OR R00971-/DCN OR RA0055-/DCN
 OR R06069/DCN OR RA0055/DCN)
 L199 0 SEA ABB=ON PLU=ON (R06069-/DRN OR R00971-/DRN OR RA0055-/DRN
 OR R06069/DRN OR RA0055/DRN)
 L200 0 SEA ABB=ON PLU=ON (R06069-/SDCE OR R00971-/SDCE OR RA0055-/SD
 CE OR R06069/SDCE OR RA0055/SDCE)
 L201 0 SEA ABB=ON PLU=ON (R06069-/SDRN OR R00971-/SDRN OR RA0055-/SD
 RN OR R06069/SDRN OR RA0055/SDRN)

FILE 'STNGUIDE' ENTERED AT 15:14:29 ON 17 JUL 2006

FILE 'USPATFULL' ENTERED AT 15:14:54 ON 17 JUL 2006

L202 1992 SEA ABB=ON PLU=ON WANG S?/AU
 L203 5 SEA ABB=ON PLU=ON SHACKELFORD R?/AU
 L204 3 SEA ABB=ON PLU=ON SHACKLEFORD R?/AU
 L205 1 SEA ABB=ON PLU=ON L202 AND (L203 OR L204)
 L206 1 SEA ABB=ON PLU=ON (L202 OR L203 OR L204) AND (L185 OR L187)

FILE 'WPIX' ENTERED AT 15:15:49 ON 17 JUL 2006

L207 0 SEA ABB=ON PLU=ON L197 AND ((L189 OR L190 OR L191))

FILE 'STNGUIDE' ENTERED AT 15:16:36 ON 17 JUL 2006
 D COST

FILE 'STNGUIDE' ENTERED AT 15:16:46 ON 17 JUL 2006

FILE 'REGISTRY' ENTERED AT 15:22:10 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 15:22:11 ON 17 JUL 2006

D QUE L77
 D QUE L78
 D QUE L182

L208 12 SEA ABB=ON PLU=ON (L77 OR L78) OR L182

FILE 'MEDLINE' ENTERED AT 15:22:15 ON 17 JUL 2006

D QUE L82
 D QUE L87
 D QUE L179

L209 13 SEA ABB=ON PLU=ON L82 OR L87 OR L179

FILE 'EMBASE' ENTERED AT 15:22:20 ON 17 JUL 2006

D QUE L127
D QUE L135
D QUE L178

L210 11 SEA ABB=ON PLU=ON L127 OR L135 OR L178

FILE 'BIOSIS' ENTERED AT 15:22:24 ON 17 JUL 2006

D QUE L154
D QUE L156
D QUE L176

L211 18 SEA ABB=ON PLU=ON L154 OR L156 OR L176

FILE 'USPATFULL' ENTERED AT 15:22:28 ON 17 JUL 2006

D QUE L205
D QUE L206

L212 1 SEA ABB=ON PLU=ON L205 OR L206

FILE 'WPIX' ENTERED AT 15:22:31 ON 17 JUL 2006

D QUE L192
D QUE L196
D QUE L207

L213 1 SEA ABB=ON PLU=ON L192 OR L196 OR L207

FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, USPATFULL, WPIX' ENTERED AT
15:23:43 ON 17 JUL 2006

L214 22 DUP REM L208 L209 L210 L211 L212 L213 (34 DUPLICATES REMOVED)
ANSWERS '1-12' FROM FILE HCAPLUS
ANSWERS '13-15' FROM FILE MEDLINE
ANSWERS '16-22' FROM FILE BIOSIS

FILE 'STNGUIDE' ENTERED AT 15:23:57 ON 17 JUL 2006

FILE 'HCAPLUS, MEDLINE, BIOSIS' ENTERED AT 15:24:47 ON 17 JUL 2006
D IBIB ABS HITIND HITSTR L214 1-12

FILE 'STNGUIDE' ENTERED AT 15:24:50 ON 17 JUL 2006

FILE 'HCAPLUS, MEDLINE, BIOSIS' ENTERED AT 15:25:01 ON 17 JUL 2006
D IALL L214 13-22

FILE 'STNGUIDE' ENTERED AT 15:25:02 ON 17 JUL 2006

FILE 'STNGUIDE' ENTERED AT 15:25:52 ON 17 JUL 2006

FILE 'HCAPLUS' ENTERED AT 15:30:14 ON 17 JUL 2006

D QUE L54
D QUE L56
D QUE L71

L215 3 SEA ABB=ON PLU=ON (L54 OR L56 OR L71) NOT L208

FILE 'MEDLINE' ENTERED AT 15:30:19 ON 17 JUL 2006

D QUE L96
D QUE L98
D QUE L105
D QUE L108
D QUE L119

L216 7 SEA ABB=ON PLU=ON (L96 OR L98 OR L105 OR L108 OR L119) NOT
L209

FILE 'EMBASE' ENTERED AT 15:30:24 ON 17 JUL 2006

D QUE L139
D QUE L142
D QUE L145
D QUE L150
L217 10 SEA ABB=ON PLU=ON (L139 OR L142 OR L145 OR L150) NOT L210
FILE 'BIOSIS' ENTERED AT 15:30:29 ON 17 JUL 2006
D QUE L168
D QUE L172
D QUE L173
L218 3 SEA ABB=ON PLU=ON (L168 OR L172 OR L173) NOT L211
FILE 'USPATFULL' ENTERED AT 15:30:33 ON 17 JUL 2006
D QUE L185
D QUE L187
L219 5 SEA ABB=ON PLU=ON (L185 OR L187) NOT L212
FILE 'WPIX' ENTERED AT 15:30:37 ON 17 JUL 2006
D QUE L197
L220 2 SEA ABB=ON PLU=ON L197 NOT L213
FILE 'STNGUIDE' ENTERED AT 15:31:04 ON 17 JUL 2006
FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, USPATFULL, WPIX' ENTERED AT
15:31:21 ON 17 JUL 2006
L221 24 DUP REM L215 L216 L217 L218 L219 L220 (6 DUPLICATES REMOVED)
ANSWERS '1-3' FROM FILE HCAPLUS
ANSWERS '4-9' FROM FILE MEDLINE
ANSWERS '10-16' FROM FILE EMBASE
ANSWER '17' FROM FILE BIOSIS
ANSWERS '18-22' FROM FILE USPATFULL
ANSWERS '23-24' FROM FILE WPIX
D IBIB ABS HITIND HITSTR L221 1-3
D IALL L221 4-17
D IBIB ABS KWIC HITSTR L221 18-22
D IALL IND L221 23-24

FILE HOME

FILE REGISTRY

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1
DICTIONARY FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

FILE STNGUIDE
FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 17, 2006 (20060717/UP).

FILE HCAPLUS

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FILE COVERS 1907 - 17 Jul 2006 VOL 145 ISS 4
FILE LAST UPDATED: 16 Jul 2006 (20060716/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE MEDLINE

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE EMBASE

FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE BIOSIS

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNS) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

FILE USPATFULL

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD)

FILE LAST UPDATED: 13 Jul 2006 (20060713/ED)

HIGHEST GRANTED PATENT NUMBER: US7076805

HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447

CA INDEXING IS CURRENT THROUGH 11 Jul 2006 (20060711/UPCA)

ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 13 Jul 2006 (20060713/PD)

REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006

USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

FILE WPIX

FILE LAST UPDATED: 14 JUL 2006 <20060714/UP>

MOST RECENT DERWENT UPDATE: 200645 <200645/DW>

DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
PLEASE VISIT:

[< http://www.stn-international.de/training_center/patents/stn_guide.pdf <](http://www.stn-international.de/training_center/patents/stn_guide.pdf)

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
<http://scientific.thomson.com/support/patents/coverage/latestupdates/>

>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ ipc_reform.html and
[<<< http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<](http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf)

>>> FOR FURTHER DETAILS ON THE FORTHCOMING DERWENT WORLD PATENTS
INDEX ENHANCEMENTS PLEASE VISIT:

[<<< http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<](http://www.stn-international.de/stndatabases/details/dwpi_r.html)

=>

=> file registry
FILE 'REGISTRY' ENTERED AT 15:22:10 ON 17 JUL 2006
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STRUCTURE FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1
DICTIONARY FILE UPDATES: 14 JUL 2006 HIGHEST RN 892755-86-1

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

=> file hcplus

FILE 'HCPLUS' ENTERED AT 15:22:11 ON 17 JUL 2006
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FILE COVERS 1907 - 17 Jul 2006 VOL 145 ISS 4
FILE LAST UPDATED: 16 Jul 2006 (20060716/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

'OBI' IS DEFAULT SEARCH FIELD FOR 'HCPLUS' FILE

=> d que L77

L74	29887 SEA FILE=HCPLUS ABB=ON	PLU=ON	WANG S?/AU
L75	49 SEA FILE=HCPLUS ABB=ON	PLU=ON	SHACKELFORD R?/AU
L76	12 SEA FILE=HCPLUS ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L77	5 SEA FILE=HCPLUS ABB=ON	PLU=ON	L74 AND (L75 OR L76)

=> d que 178

L42	1665	SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/OBI
L43	2356	SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BI
L44	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/OBI
L45	8	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/BI
L46	8	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS-BAR/BI
L47	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
L48	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOT?/BI
L49	2363	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(ATAXIA (2A) TELANGIECT?)/BI
L52	2369	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49)
L74	29887	SEA FILE=HCAPLUS ABB=ON	PLU=ON	WANG S?/AU
L75	49	SEA FILE=HCAPLUS ABB=ON	PLU=ON	SHACKELFORD R?/AU
L76	12	SEA FILE=HCAPLUS ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L78	12	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L52 AND (L74 OR L75 OR L76)

=> d que L182

L1	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6	SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
L12	0	SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATE"/CN OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0	SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6	SEA FILE=REGISTRY ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4	SEA FILE=REGISTRY ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI C?/CN
L23	23	SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	70-51-9
L31	24	SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L37	22	SEA FILE=REGISTRY ABB=ON	PLU=ON	(117-39-5/BI OR 146426-40-6/B I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR 520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
L38	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	50-78-2
L39	21	SEA FILE=REGISTRY ABB=ON	PLU=ON	L37 NOT L38

L42	1665	SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/OBI
L43	2356	SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BI
L44	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/OBI
L45	8	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/BI
L46	8	SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS-BAR/BI
L47	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
L48	0	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOT?/BI
L49	2363	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(ATAXIA (2A) TELANGIECT?) /BI
L50	15362	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CHELATING AGENTS+OLD,NT/CT
L51	40989	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L31
L52	2369	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49)
L53	52491	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L50 OR L51)
L54	7	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L52 AND L53
L55	34358	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L39
L56	4	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L54 AND L55
L68	15322	SEA FILE=HCAPLUS ABB=ON	PLU=ON	HYDROXAMIC ACIDS+NT/CT
L69	29	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L68 AND L52
L70	132130	SEA FILE=HCAPLUS ABB=ON	PLU=ON	CHELAT?/BI
L71	3	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L69 AND L70
L74	29887	SEA FILE=HCAPLUS ABB=ON	PLU=ON	WANG S?/AU
L75	49	SEA FILE=HCAPLUS ABB=ON	PLU=ON	SHACKELFORD R?/AU
L76	12	SEA FILE=HCAPLUS ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L77	5	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L74 AND (L75 OR L76)
L78	12	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L52 AND (L74 OR L75 OR L76)
L180	12	SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L77 OR L78)
L181	7	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L54 OR L56 OR L71
L182	4	SEA FILE=HCAPLUS ABB=ON	PLU=ON	L180 AND L181

=> s L77-L78 or L182

L208 12 (L77 OR L78) OR L182

=> file medline

FILE 'MEDLINE' ENTERED AT 15:22:15 ON 17 JUL 2006

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que L82

L79	9295 SEA FILE=MEDLINE ABB=ON	PLU=ON	WANG S?/AU
L80	81 SEA FILE=MEDLINE ABB=ON	PLU=ON	SHACKELFORD R?/AU
L81	10 SEA FILE=MEDLINE ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L82	3 SEA FILE=MEDLINE ABB=ON	PLU=ON	L79 AND (L80 OR L81)

=> d que L87

L79	9295 SEA FILE=MEDLINE ABB=ON	PLU=ON	WANG S?/AU
L80	81 SEA FILE=MEDLINE ABB=ON	PLU=ON	SHACKELFORD R?/AU
L81	10 SEA FILE=MEDLINE ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L83	2457 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/CT
L84	3932 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L85	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA (2A) TELANGIECTASIA
L86	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	(L83 OR L84 OR L85)
L87	13 SEA FILE=MEDLINE ABB=ON	PLU=ON	L86 AND (L79 OR L80 OR L81)

=> d que l179

L1	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATED/CN
L12	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATED" /CN OR "DEFEROXAMINE METHANESULFONATE" /CN)
L14	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4 SEA FILE=REGISTRY ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI C?/CN
L23	23 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	70-51-9
L31	24 SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L37	22 SEA FILE=REGISTRY ABB=ON	PLU=ON	(117-39-5/BI OR 146426-40-6/B I OR 153-18-4/BI OR 17912-87-7/B I OR 446-72-0/B I OR 480-16-0/B I OR 480-18-2/B I OR 480-40-0/B I OR 480-41-1/B I OR 482-39-3/B I OR 490-46-0/B I OR 491-70-3/B I OR 491-80-5/B I OR 50-78-2/B I OR 520-26-3/B I OR 520-27-4/B I OR 520-33-2/B I OR 520-36-5/B I OR 522-12-3/B I OR 525-82-6/B I OR 577-85-5/B I OR 989-51-5/B)
L38	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	50-78-2
L39	21 SEA FILE=REGISTRY ABB=ON	PLU=ON	L37 NOT L38

L79 9295 SEA FILE=MEDLINE ABB=ON PLU=ON WANG S?/AU
 L80 81 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKELFORD R?/AU
 L81 10 SEA FILE=MEDLINE ABB=ON PLU=ON SHACKLEFORD R?/AU
 L82 3 SEA FILE=MEDLINE ABB=ON PLU=ON L79 AND (L80 OR L81)
 L83 2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
 L84 3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L85 3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
 L86 3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
 L87 13 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L79 OR L80 OR L81)
 L88 13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT
 L89 92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT
 L90 3231 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS/CT
 L91 19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
 L92 1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT
 L93 6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT
 L94 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L95 68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
 L96 7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
 OR L91 OR L92 OR L93) OR L95)
 L97 37091 SEA FILE=MEDLINE ABB=ON PLU=ON CHELAT?
 L98 5 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND L97
 L100 61053 SEA FILE=MEDLINE ABB=ON PLU=ON ANTIOXID?
 L101 18939 SEA FILE=MEDLINE ABB=ON PLU=ON FLAV!NOID?/BI
 L102 32882 SEA FILE=MEDLINE ABB=ON PLU=ON FLAVONOIDS+NT/CT
 L103 QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
 L105 3 SEA FILE=MEDLINE ABB=ON PLU=ON L96 AND ((L100 OR L101 OR
 L102 OR L103))
 L106 SEL PLU=ON L39 1- CHEM : 344 TERMS
 L107 19788 SEA FILE=MEDLINE ABB=ON PLU=ON L106
 L108 2 SEA FILE=MEDLINE ABB=ON PLU=ON L107 AND L96
 L109 QUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESF
 ERROXAMIN? OR DEFERRIOXAMIN?
 L110 QUE ABB=ON PLU=ON EDETIC ACID/CT
 L111 QUE ABB=ON PLU=ON CP94
 L112 QUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
 L113 QUE ABB=ON PLU=ON APOFERRITIN/CT
 L114 QUE ABB=ON PLU=ON CDTA
 L115 QUE ABB=ON PLU=ON DTPA OR PENTATIC ACID
 L116 QUE ABB=ON PLU=ON PENICILLAMINE
 L117 QUE ABB=ON PLU=ON BATHOCUPROINE
 L118 QUE ABB=ON PLU=ON BATHOCUPROIN
 L119 6 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111
 OR L112 OR L113 OR L114 OR L115 OR L116 OR L117 OR L118)
 L120 1 SEA FILE=MEDLINE ABB=ON PLU=ON L119 AND L107
 L121 13 SEA FILE=MEDLINE ABB=ON PLU=ON L82 OR L87
 L122 10 SEA FILE=MEDLINE ABB=ON PLU=ON L96 OR L98 OR L105 OR L108 OR
 L119 OR L120
 L179 3 SEA FILE=MEDLINE ABB=ON PLU=ON L121 AND L122

=> s L82 or L87 or L179

L209 13 L82 OR L87 OR L179

=> file embase

FILE 'EMBASE' ENTERED AT 15:22:20 ON 17 JUL 2006
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FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que L127

L124	7035 SEA FILE=EMBASE ABB=ON	PLU=ON	WANG S?/AU
L125	5 SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L126	27 SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKELFORD R?/AU
L127	4 SEA FILE=EMBASE ABB=ON	PLU=ON	L124 AND (L125 OR L126)

=> d que L135

L124	7035 SEA FILE=EMBASE ABB=ON	PLU=ON	WANG S?/AU
L125	5 SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L126	27 SEA FILE=EMBASE ABB=ON	PLU=ON	SHACKELFORD R?/AU
L128	2332 SEA FILE=EMBASE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA+UF/CT
L129	3044 SEA FILE=EMBASE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L130	62 SEA FILE=EMBASE ABB=ON	PLU=ON	LOUIS BAR
L131	2 SEA FILE=EMBASE ABB=ON	PLU=ON	ATAXIA TELANGIECTATICA
L132	0 SEA FILE=EMBASE ABB=ON	PLU=ON	TELANGIECTASIA CEREBELLOOCULOCUTANEA
L133	0 SEA FILE=EMBASE ABB=ON	PLU=ON	TELANGIECTASIA CEREBELLO
	OCULOCUTANEA		
L134	3053 SEA FILE=EMBASE ABB=ON	PLU=ON	(L128 OR L129 OR L130 OR L131 OR L132 OR L133)
L135	11 SEA FILE=EMBASE ABB=ON	PLU=ON	(L124 OR L125 OR L126) AND L134

=> d que L178

L1	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATED/CN
L12	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATED"/CN OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN

L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/B1 OR 146426-40-6/B
 I OR 153-18-4/B1 OR 17912-87-7/B1 OR 446-72-0/B1 OR 480-16-0/B1
 OR 480-18-2/B1 OR 480-40-0/B1 OR 480-41-1/B1 OR 482-39-3/B1
 OR 490-46-0/B1 OR 491-70-3/B1 OR 491-80-5/B1 OR 50-78-2/B1 OR
 520-26-3/B1 OR 520-27-4/B1 OR 520-33-2/B1 OR 520-36-5/B1 OR
 522-12-3/B1 OR 525-82-6/B1 OR 577-85-5/B1 OR 989-51-5/B1)
 L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
 L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
 L124 7035 SEA FILE=EMBASE ABB=ON PLU=ON WANG S?/AU
 L125 5 SEA FILE=EMBASE ABB=ON PLU=ON SHACKLEFORD R?/AU
 L126 27 SEA FILE=EMBASE ABB=ON PLU=ON SHACKELFORD R?/AU
 L127 4 SEA FILE=EMBASE ABB=ON PLU=ON L124 AND (L125 OR L126)
 L128 2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
 L129 3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L130 62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
 L131 2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
 L132 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
 TANEA
 L133 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
 OCULOCUTANEA
 L134 3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
 OR L132 OR L133)
 L135 11 SEA FILE=EMBASE ABB=ON PLU=ON (L124 OR L125 OR L126) AND
 L134
 L136 98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
 L137 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L138 61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
 L139 14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
 L140 25033 SEA FILE=EMBASE ABB=ON PLU=ON FLAVONOIDS+NT/CT
 L141 35447 SEA FILE=EMBASE ABB=ON PLU=ON ANTIOXIDANT+NT/CT
 L142 4 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND (L140 OR L141)
 L143 SEL PLU=ON L39 1- CHEM : 344 TERMS
 L144 24101 SEA FILE=EMBASE ABB=ON PLU=ON L143
 L145 2 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND L144
 L146 14 SEA FILE=EMBASE ABB=ON PLU=ON L139 OR L142 OR L145
 L147 11 SEA FILE=EMBASE ABB=ON PLU=ON L127 OR L135
 L149 31221 SEA FILE=EMBASE ABB=ON PLU=ON CHELAT?
 L150 4 SEA FILE=EMBASE ABB=ON PLU=ON L149 AND L134
 L177 14 SEA FILE=EMBASE ABB=ON PLU=ON L146 OR L150
 L178 4 SEA FILE=EMBASE ABB=ON PLU=ON L147 AND L177

=> s L127 or L135 or L178

L210 11 L127 OR L135 OR L178

=> file biosis

FILE 'BIOSIS' ENTERED AT 15:22:24 ON 17 JUL 2006
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FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNS) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

=> d que L154

L151	10521	SEA FILE=BIOSIS ABB=ON	PLU=ON	WANG S?/AU
L152	52	SEA FILE=BIOSIS ABB=ON	PLU=ON	SHACKELFORD R?/AU
L153	7	SEA FILE=BIOSIS ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L154	6	SEA FILE=BIOSIS ABB=ON	PLU=ON	L151 AND (L152 OR L153)

=> d que L156

L151	10521	SEA FILE=BIOSIS ABB=ON	PLU=ON	WANG S?/AU
L152	52	SEA FILE=BIOSIS ABB=ON	PLU=ON	SHACKELFORD R?/AU
L153	7	SEA FILE=BIOSIS ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L155	3180	SEA FILE=BIOSIS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L156	18	SEA FILE=BIOSIS ABB=ON	PLU=ON	(L151 OR L152 OR L153) AND L155

=> d que L176

L1	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2	SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6	SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
L12	0	SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATE"/CN OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0	SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6	SEA FILE=REGISTRY ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4	SEA FILE=REGISTRY ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI C?/CN
L23	23	SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29	1	SEA FILE=REGISTRY ABB=ON	PLU=ON	70-51-9
L31	24	SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L37	22	SEA FILE=REGISTRY ABB=ON	PLU=ON	(117-39-5/BI OR 146426-40-6/B I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR

520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
 522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)

L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
 L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
 L151 10521 SEA FILE=BIOSIS ABB=ON PLU=ON WANG S?/AU
 L152 52 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKELFORD R?/AU
 L153 7 SEA FILE=BIOSIS ABB=ON PLU=ON SHACKLEFORD R?/AU
 L154 6 SEA FILE=BIOSIS ABB=ON PLU=ON L151 AND (L152 OR L153)
 L155 3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L156 18 SEA FILE=BIOSIS ABB=ON PLU=ON (L151 OR L152 OR L153) AND
 L155
 L157 38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L159 59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
 L160 3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
 L161 4049 SEA FILE=BIOSIS ABB=ON PLU=ON TRANSITION METAL?
 L162 160 SEA FILE=BIOSIS ABB=ON PLU=ON TRANSITION ELEM?
 L163 3396 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROPHOR?
 L164 42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
 L165 72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
 L166 3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
 QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
 L168 7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167
 L170 SEL PLU=ON L39 1- CHEM : 344 TERMS
 L171 31206 SEA FILE=BIOSIS ABB=ON PLU=ON L170
 L172 3 SEA FILE=BIOSIS ABB=ON PLU=ON L171 AND L168
 L173 0 SEA FILE=BIOSIS ABB=ON PLU=ON L168 AND (L161 OR L162)
 L174 18 SEA FILE=BIOSIS ABB=ON PLU=ON L154 OR L156
 L175 7 SEA FILE=BIOSIS ABB=ON PLU=ON L168 OR L172 OR L173
 L176 4 SEA FILE=BIOSIS ABB=ON PLU=ON L174 AND L175

=> s L154 or L156 or L176

L211 18 L154 OR L156 OR L176

=> file uspatfull

FILE 'USPATFULL' ENTERED AT 15:22:28 ON 17 JUL 2006
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD)
 FILE LAST UPDATED: 13 Jul 2006 (20060713/ED)
 HIGHEST GRANTED PATENT NUMBER: US7076805
 HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447
 CA INDEXING IS CURRENT THROUGH 11 Jul 2006 (20060711/UPCA)
 ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 13 Jul 2006 (20060713/PD)
 REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006
 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

=> d que L205

L202 1992 SEA FILE=USPATFULL ABB=ON PLU=ON WANG S?/AU
 L203 5 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKELFORD R?/AU
 L204 3 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKLEFORD R?/AU
 L205 1 SEA FILE=USPATFULL ABB=ON PLU=ON L202 AND (L203 OR L204)

=> d que L206

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
 L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
 L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
 L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
 L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
 L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
 L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6)
 L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
 L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
 L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
 L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
 L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/B1 OR 146426-40-6/B
 I OR 153-18-4/B1 OR 17912-87-7/B1 OR 446-72-0/B1 OR 480-16-0/B1
 OR 480-18-2/B1 OR 480-40-0/B1 OR 480-41-1/B1 OR 482-39-3/B1
 OR 490-46-0/B1 OR 491-70-3/B1 OR 491-80-5/B1 OR 50-78-2/B1 OR
 520-26-3/B1 OR 520-27-4/B1 OR 520-33-2/B1 OR 520-36-5/B1 OR
 522-12-3/B1 OR 525-82-6/B1 OR 577-85-5/B1 OR 989-51-5/B1)
 L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
 L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
 L183 5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31
 L184 2355 SEA FILE=USPATFULL ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L185 6 SEA FILE=USPATFULL ABB=ON PLU=ON L183 AND L184
 L186 1795 SEA FILE=USPATFULL ABB=ON PLU=ON L39
 L187 1 SEA FILE=USPATFULL ABB=ON PLU=ON L185 AND L186
 L202 1992 SEA FILE=USPATFULL ABB=ON PLU=ON WANG S?/AU
 L203 5 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKELFORD R?/AU
 L204 3 SEA FILE=USPATFULL ABB=ON PLU=ON SHACKLEFORD R?/AU
 L206 1 SEA FILE=USPATFULL ABB=ON PLU=ON (L202 OR L203 OR L204) AND
 (L185 OR L187)

=> s L205 or L206

L212 1 L205 OR L206

=> file wpix

FILE 'WPIX' ENTERED AT 15:22:31 ON 17 JUL 2006
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FILE LAST UPDATED: 14 JUL 2006 <20060714/UP>
 MOST RECENT DERWENT UPDATE: 200645 <200645/DW>
 DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
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>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
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>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ ipc_reform.html and
[<<<http://scientific.thomson.com/media/scpdf/ ipcrdwpi.pdf><<<](http://scientific.thomson.com/media/scpdf/ ipcrdwpi.pdf)

>>> FOR FURTHER DETAILS ON THE FORTHCOMING DERWENT WORLD PATENTS
 INDEX ENHANCEMENTS PLEASE VISIT:
[<<<http://www.stn-international.de/stndatabases/details/dwpi_r.html><<<](http://www.stn-international.de/stndatabases/details/dwpi_r.html)
 'BIX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE

=> d que L192

L189	5402	SEA FILE=WPIX ABB=ON	PLU=ON	WANG S?/AU
L190	0	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L191	3	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKELFORD R?/AU
L192	1	SEA FILE=WPIX ABB=ON	PLU=ON	L189 AND (L190 OR L191)

=> d que L196

L188	247	SEA FILE=WPIX ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BIX
L189	5402	SEA FILE=WPIX ABB=ON	PLU=ON	WANG S?/AU
L190	0	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L191	3	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKELFORD R?/AU
L196	0	SEA FILE=WPIX ABB=ON	PLU=ON	L188 AND (L189 OR L190 OR L191)

=> d que L207

L188	247	SEA FILE=WPIX ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BIX
L189	5402	SEA FILE=WPIX ABB=ON	PLU=ON	WANG S?/AU
L190	0	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKLEFORD R?/AU
L191	3	SEA FILE=WPIX ABB=ON	PLU=ON	SHACKELFORD R?/AU
L195	5533	SEA FILE=WPIX ABB=ON	PLU=ON	(RAAMBT/DCN OR RAGNQ8/DCN OR RA0DFA/DCN OR RA0EMC/DCN OR RA0JBK/DCN OR RA00TF/DCN OR RA0055/DCN OR RA021P/DCN OR RA0529/DCN OR RA1HHQ/DCN OR RA1XA5/DCN OR RA37W9/DCN OR R00064/DCN OR R00195/DCN OR R00268/DCN OR R00971/DCN OR R01179/DCN OR R01318/DCN OR R01319/DCN OR R03811/DCN OR R03812/DCN OR R03949/DCN OR R04870/DCN OR R06069/DCN OR R06174/DCN OR R06413/DCN OR R06747/DCN OR R07001/DCN OR R07027/DCN OR R08105/DCN OR R08504/DCN OR R09163/DCN OR R09222/DCN OR R09884/DCN OR R11605/DCN OR R19085/DCN OR R19452/DCN OR R20811/DCN OR R22037/DCN)
L197	2	SEA FILE=WPIX ABB=ON	PLU=ON	L188 AND L195
L207	0	SEA FILE=WPIX ABB=ON	PLU=ON	L197 AND ((L189 OR L190 OR L191))

=> s L192 or L196 or L207

L213 1 L192 OR L196 OR L207

=> dup rem L208 L209 L210 L211 L212 L213
FILE 'HCAPLUS' ENTERED AT 15:23:43 ON 17 JUL 2006
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PROCESSING COMPLETED FOR L208

PROCESSING COMPLETED FOR L209

PROCESSING COMPLETED FOR L210

PROCESSING COMPLETED FOR L211

PROCESSING COMPLETED FOR L212

PROCESSING COMPLETED FOR L213

L214 22 DUP REM L208 L209 L210 L211 L212 L213 (34 DUPLICATES REMOVED)
ANSWERS '1-12' FROM FILE HCAPLUS
ANSWERS '13-15' FROM FILE MEDLINE
ANSWERS '16-22' FROM FILE BIOSIS

=> => d ibib abs hitind hitstr L214 1-12; d iall L214 13-22
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS, MEDLINE, BIOSIS' - CONTINUE? (Y)/N:y

L214 ANSWER 1 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2005:34588 HCAPLUS

DOCUMENT NUMBER: 142:127600

TITLE: Methods and compositions using *chelating*
agents for treatment of *ataxia-telangiectasia* and diseases associated with
oxidative stress and genomic instability

INVENTOR(S): Wang, Suming; Shackelford, Rodney E.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 37 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005009760	A1	20050113	US 2003-617943	20030711
PRIORITY APPLN. INFO.:			US 2003-617943	20030711
AB This invention relates to the methods and pharmaceutical compns. for treating diseases or disorders associated with oxidative stress and/or				

genomic instability. In particular, the invention relates to methods for treating **ataxia telangiectasia** (AT) and such disease states by administering a therapeutically effective amount of a **chelating** agent to increase genomic stability and/or decrease oxidative stress. The ferrous iron **chelating** agent desferrioxamine was used to increase the genomic stability of **ataxia telangiectasia** cells.

- IC ICM A61K031-7048
 ICS A61K031-353; A61K031-198; A61K031-195
 INCL 514027000; 514456000; 514566000
 CC 1-10 (Pharmacology)
 Section cross-reference(s): 63
- ST ataxia telangiectasia treatment **chelating** agent; oxidative stress disease treatment **chelating** agent; genomic instability disease treatment **chelating** agent; desferrioxamine treatment **ataxia telangiectasia**
- IT Alkenes, biological studies
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (alkatrienes, as **chelating** agent; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Ferritins
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (apoferritins, as **chelating** agent; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Flavonoids
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (as antioxidant; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT **Hydroxamic acids**
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (as **chelating** agent, tri-,intermediates; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Disease, animal
 (associated with oxidative stress or genomic instability; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Nervous system, disease
 (**ataxia telangiectasia**; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Cell membrane
 (**chelating** agent capable of crossing; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Transition metals, biological studies
 RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
 (**chelating** agents binding; **chelating** agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)
- IT Animals

Chelating agents

Combination chemotherapy

Drug delivery systems

Human

Oxidative stress, biological

(chelating agents for treatment of **ataxia**)**telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT Antioxidants

(further treatment with; chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT Gene, animal

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)

(instability; chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT 117-39-5, Quercetin 153-18-4, Rutin 446-72-0,

Genistein 480-16-0, Morin 480-18-2, Taxifolin

480-40-0, Chrysin 480-41-1, Naringenin 482-39-3

, Afzelin 490-46-0, Epicatechin 491-70-3, Luteolin

491-80-5, Biochanin A 520-26-3, Hesperidin

520-27-4, Diosmin 520-33-2, Hesperetin 520-36-5

, Apigenin 522-12-3, Quercitrin 525-82-6, Flavone

577-85-5, Flavonol 989-51-5, Epigallocatechin gallate

17912-87-7, Myricitrin 146426-40-6, Flavopiridol

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(as antioxidant; chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT 52-67-5, Penicillamine 60-00-4, EDTA, biological studies

67-43-6, Diethylenetriamine-N,N,N',N'',N'''-pentaacetic acid

70-51-9D, Desferrioxamine, compds. 138-14-7, Desferal

138-14-7D, Desferrioxamine B mesylate, compds. 13291-61-7

, trans-1,2-Diaminocyclohexane-N,N,N',N'-tetraacetic acid

14836-73-8, Ferrioxamine 73348-75-1 115900-75-9

, CP94

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(as chelating agent; chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT 7439-89-6, Iron, biological studies 7440-50-8, Copper, biological studies

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)

(chelating agent of; chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)IT 75-91-2, tert-Butyl hydroperoxide 7447-39-4, Copper chloride (CuCl₂), biological studies 7758-94-3, Ferrous chloride

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)

(chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT 50-78-2, Aspirin 70-51-9, Desferrioxamine

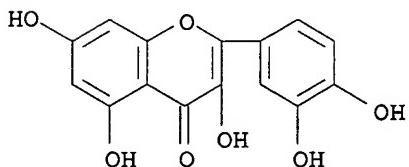
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);

THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (chelating agents for treatment of **ataxia telangiectasia** and diseases associated with oxidative stress and genomic instability)

IT 117-39-5, Quercetin 153-18-4, Rutin 446-72-0,
 Genistein 480-16-0, Morin 480-18-2, Taxifolin
 480-40-0, Chrysin 480-41-1, Naringenin 482-39-3
 , Afzelin 490-46-0, Epicatechin 491-70-3, Luteolin
 491-80-5, Biochanin A 520-26-3, Hesperidin
 520-27-4, Diosmin 520-33-2, Hesperetin 520-36-5
 , Apigenin 522-12-3, Quercitrin 525-82-6, Flavone
 577-85-5, Flavonol 989-51-5, Epigallocatechin gallate
 17912-87-7, Myricitrin 146426-40-6, Flavopiridol
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
 THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (as antioxidant; chelating agents for treatment of
ataxia telangiectasia and diseases associated with
 oxidative stress and genomic instability)

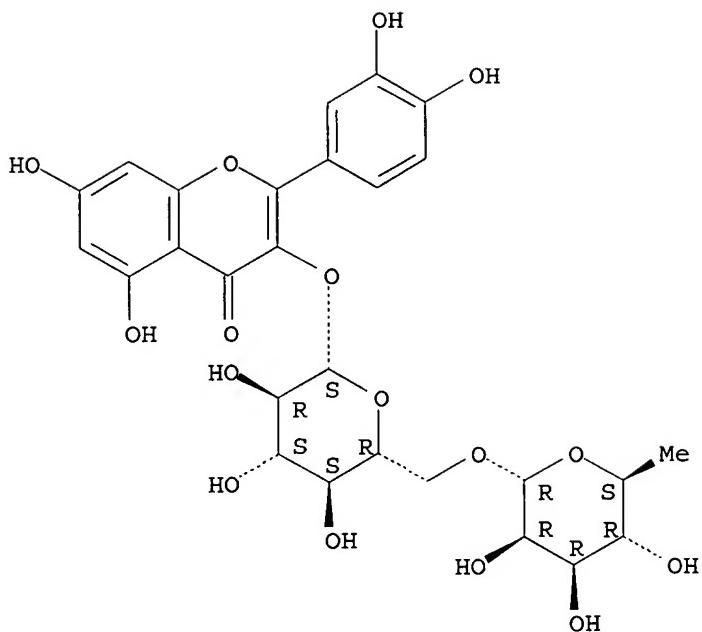
RN 117-39-5 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI)
 (CA INDEX NAME)

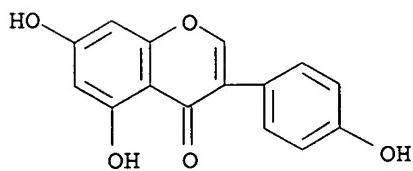


RN 153-18-4 HCAPLUS
 CN 4H-1-Benzopyran-4-one, 3-[[6-O-(6-deoxy- α -L-mannopyranosyl)- β -D-glucopyranosyl]oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA INDEX NAME)

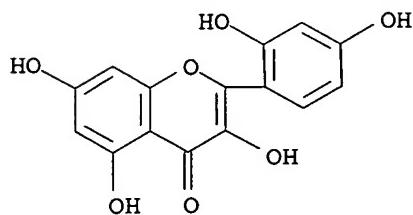
Absolute stereochemistry. Rotation (+).



RN 446-72-0 HCAPLUS
 CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-3-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)

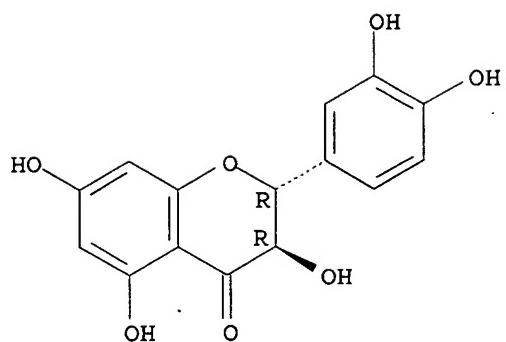


RN 480-16-0 HCAPLUS
 CN 4H-1-Benzopyran-4-one, 2-(2,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI)
 (CA INDEX NAME)



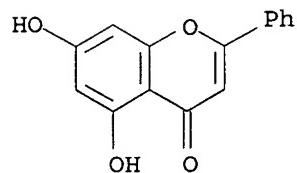
RN 480-18-2 HCAPLUS
 CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-2,3-dihydro-3,5,7-trihydroxy-, (2R,3R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



RN 480-40-0 HCAPLUS

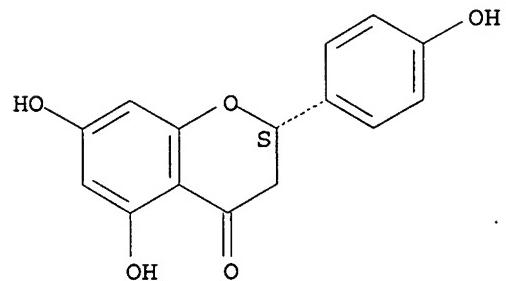
CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-2-phenyl- (9CI) (CA INDEX NAME)



RN 480-41-1 HCAPLUS

CN 4H-1-Benzopyran-4-one, 2,3-dihydro-5,7-dihydroxy-2-(4-hydroxyphenyl)-, (2S)- (9CI) (CA INDEX NAME)

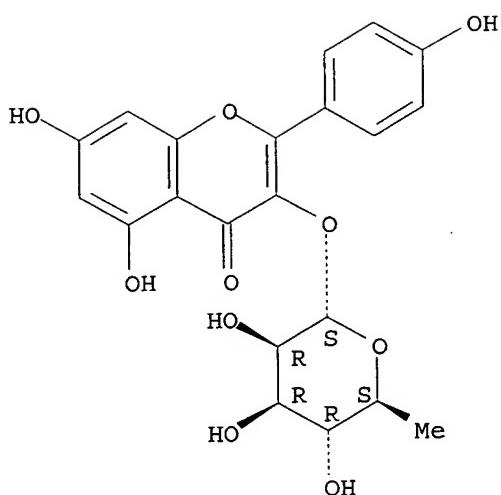
Absolute stereochemistry.



RN 482-39-3 HCAPLUS

CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy-alpha-L-mannopyranosyl)oxy]-5,7-dihydroxy-2-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)

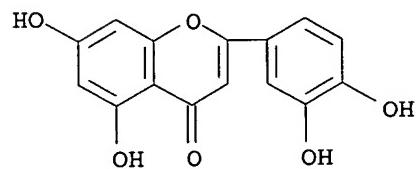
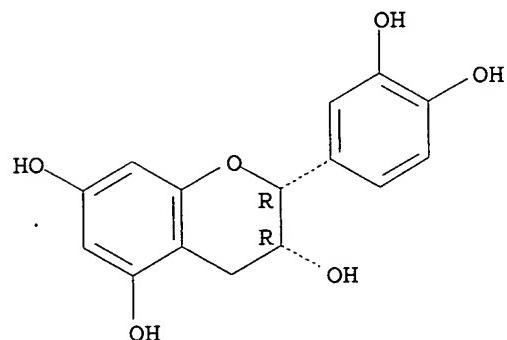
Absolute stereochemistry. Rotation (-).

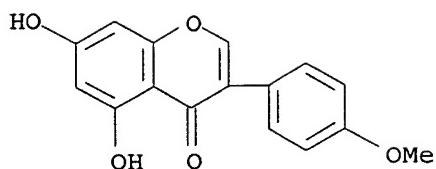


RN 490-46-0 HCAPLUS

CN 2H-1-Benzopyran-3,5,7-triol, 2-(3,4-dihydroxyphenyl)-3,4-dihydro-,
(2R,3R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

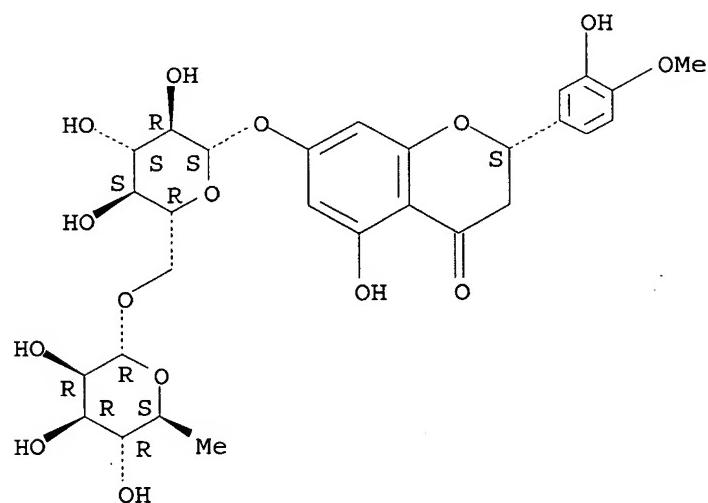
RN 491-80-5 HCAPLUS
CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-3-(4-methoxyphenyl)- (9CI) (CA INDEX NAME)



RN 520-26-3 HCAPLUS

CN 4H-1-Benzopyran-4-one, 7-[[6-O-(6-deoxy-alpha-L-mannopyranosyl)-beta-D-glucopyranosyl]oxy]-2,3-dihydro-5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)-(2S)-(9CI) (CA INDEX NAME)

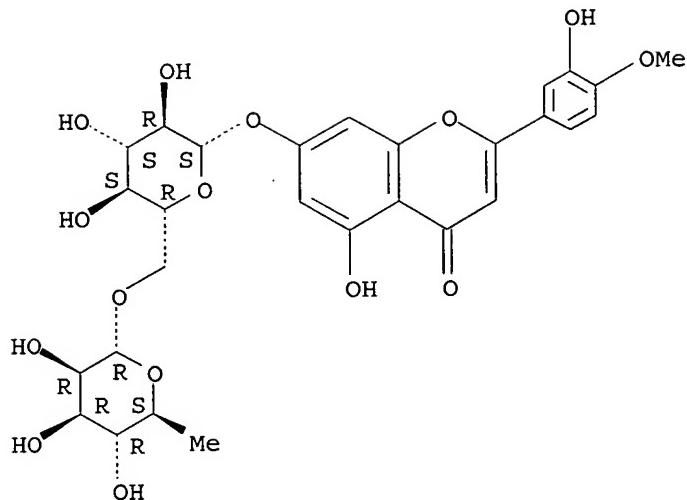
Absolute stereochemistry.



RN 520-27-4 HCAPLUS

CN 4H-1-Benzopyran-4-one, 7-[[6-O-(6-deoxy-alpha-L-mannopyranosyl)-beta-D-glucopyranosyl]oxy]-5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)-(9CI) (CA INDEX NAME)

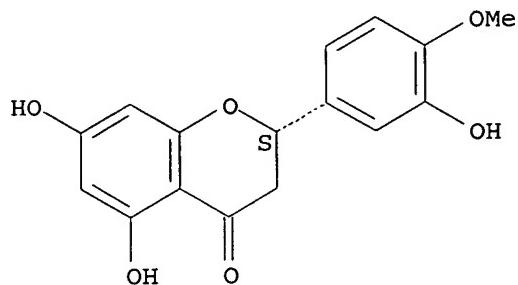
Absolute stereochemistry.



RN 520-33-2 HCAPLUS

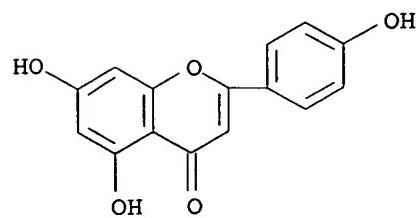
CN 4H-1-Benzopyran-4-one, 2,3-dihydro-5,7-dihydroxy-2-(3-hydroxy-4-methoxyphenyl)-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 520-36-5 HCAPLUS

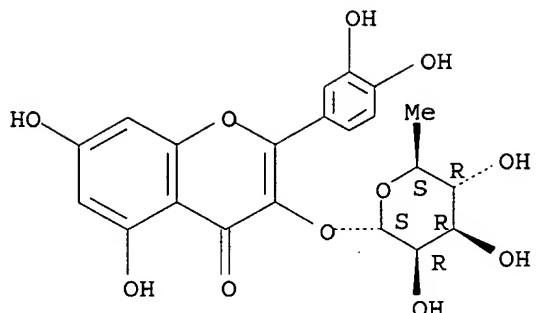
CN 4H-1-Benzopyran-4-one, 5,7-dihydroxy-2-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)



RN 522-12-3 HCAPLUS

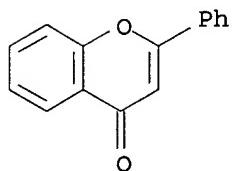
CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy- α -L-mannopyranosyl)oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



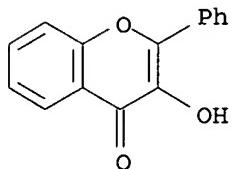
RN 525-82-6 HCPLUS

CN 4H-1-Benzopyran-4-one, 2-phenyl- (9CI) (CA INDEX NAME)



RN 577-85-5 HCPLUS

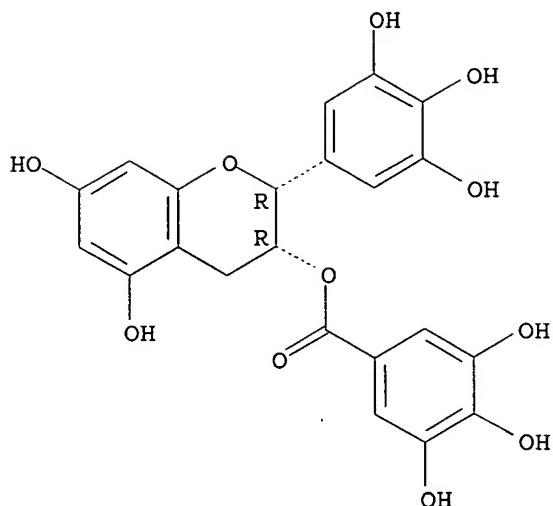
CN 4H-1-Benzopyran-4-one, 3-hydroxy-2-phenyl- (9CI) (CA INDEX NAME)



RN 989-51-5 HCPLUS

CN Benzoic acid, 3,4,5-trihydroxy-, (2R,3R)-3,4-dihydro-5,7-dihydroxy-2-(3,4,5-trihydroxyphenyl)-2H-1-benzopyran-3-yl ester (9CI) (CA INDEX NAME)

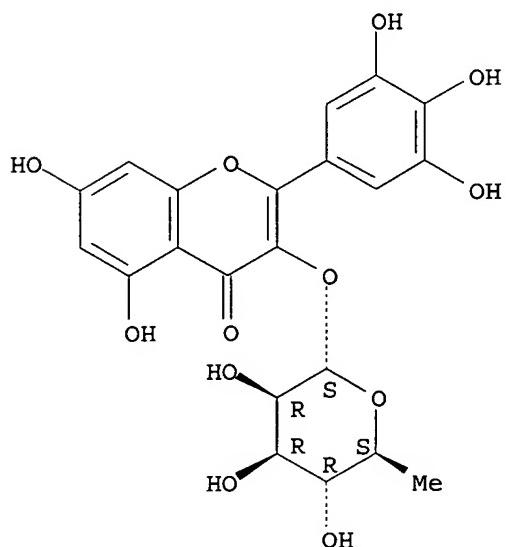
Absolute stereochemistry. Rotation (-).



RN 17912-87-7 HCAPLUS

CN 4H-1-Benzopyran-4-one, 3-[(6-deoxy-alpha-L-mannopyranosyl)oxy]-5,7-dihydroxy-2-(3,4,5-trihydroxyphenyl)- (9CI) (CA INDEX NAME)

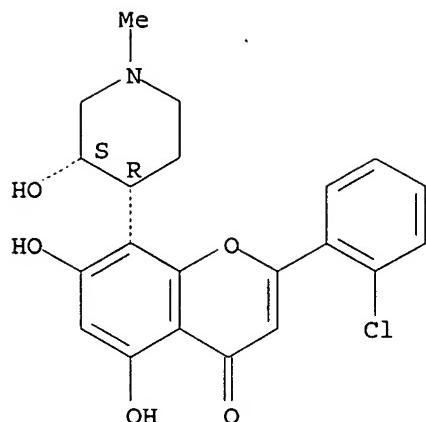
Absolute stereochemistry. Rotation (-).



RN 146426-40-6 HCAPLUS

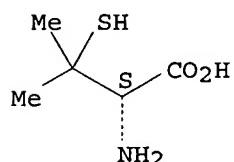
CN 4H-1-Benzopyran-4-one, 2-(2-chlorophenyl)-5,7-dihydroxy-8-[(3S,4R)-3-hydroxy-1-methyl-4-piperidinyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

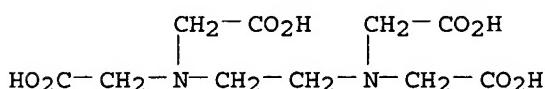


IT 52-67-5, Penicillamine 60-00-4, EDTA, biological studies
 67-43-6, Diethylenetriamine-N,N,N',N'',N'''-pentaacetic acid
 70-51-9D, Desferrioxamine, compds. 138-14-7, Desferal
 138-14-7D, Desferrioxamine B mesylate, compds. 13291-61-7
 , trans-1,2-Diaminocyclohexane-N,N,N',N'-tetraacetic acid
 14836-73-8, Ferrioxamine 73348-75-1 115900-75-9
 , CP94
 RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
 THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (as chelating agent; chelating agents for treatment
 of ataxia telangiectasia and diseases associated with
 oxidative stress and genomic instability)
 RN 52-67-5 HCAPLUS
 CN D-Valine, 3-mercaptop- (9CI) (CA INDEX NAME)

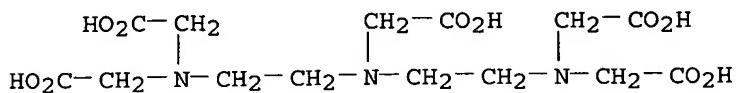
Absolute stereochemistry.



RN 60-00-4 HCAPLUS
 CN Glycine, N,N'-1,2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME)]



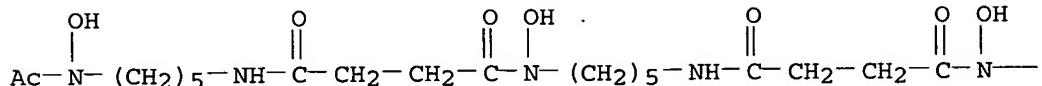
RN 67-43-6 HCAPLUS
 CN Glycine, N,N-bis[2-[bis(carboxymethyl)amino]ethyl]- (7CI, 8CI, 9CI) (CA INDEX NAME)



RN 70-51-9 HCPLUS

CN Butanediamide, N' - [5 - [4 - [[5 - (acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl -N - (5 - aminopentyl) -N - hydroxy - (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B

--- (CH_2)₅-NH₂

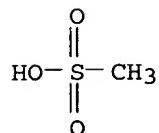
RN 138-14-7 HCPLUS

CN Butanediamide, N' - [5 - [4 - [[5 - (acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl -N - (5 - aminopentyl) -N - hydroxy - , monomethanesulfonate (salt) (9CI) (CA INDEX NAME)

CM 1

CRN 75-75-2

CMF C H4 O3 S

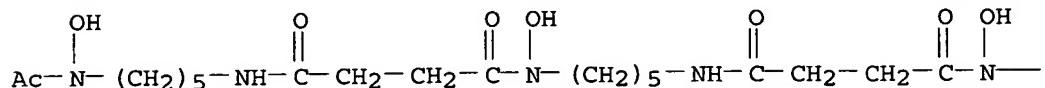


CM 2

CRN 70-51-9

CMF C25 H48 N6 O8

PAGE 1-A



PAGE 1-B

 $\text{---} (\text{CH}_2)_5 \text{--- NH}_2$

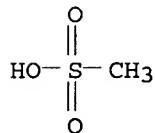
RN 138-14-7 HCPLUS

CN Butanediamide, N' - [5 - [4 - [5 - (acetylhydroxyamino)pentyl]amino] -1,4-dioxobutyl]hydroxyamino]pentyl] -N - (5 - aminopentyl) -N - hydroxy-, monomethanesulfonate (salt) (9CI) (CA INDEX NAME)

CM 1

CRN 75-75-2

CMF C H4 O3 S

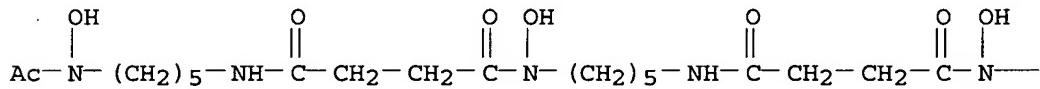


CM 2

CRN 70-51-9

CMF C25 H48 N6 O8

PAGE 1-A



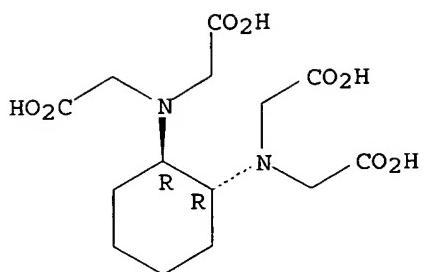
PAGE 1-B

 $\text{---} (\text{CH}_2)_5 \text{--- NH}_2$

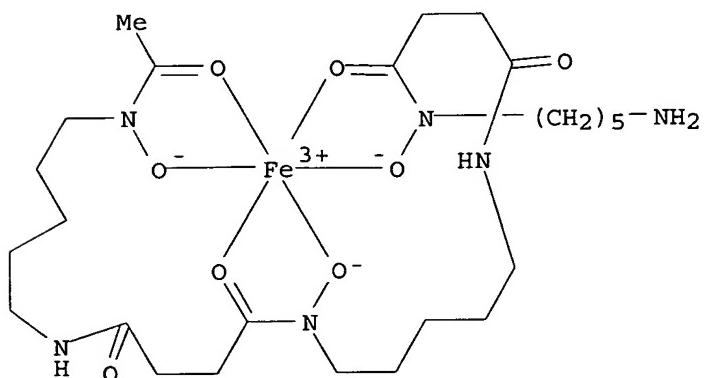
RN 13291-61-7 HCPLUS

CN Glycine, N,N'-(1R,2R)-1,2-cyclohexanediylbis[N-(carboxymethyl)-, rel-(9CI) (CA INDEX NAME)

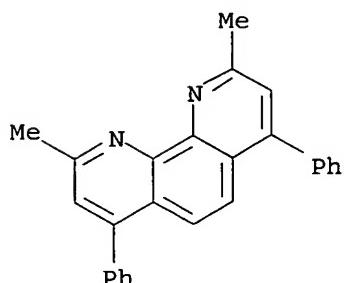
Relative stereochemistry.



RN 14836-73-8 HCAPLUS
 CN Iron, [N' - {5 - [{4 - [{5 - [(acetyl- κ O) (hydroxy- κ O) amino]pentyl} amino] -1 - (oxo- κ O) -4 -oxobutyl} (hydroxy- κ O) amino]pentyl] -N - (5 - aminopentyl) -N - (hydroxy- κ O) butanediamidato(3-) - κ O1] -, (OC-6-64) - (9CI) (CA INDEX NAME)



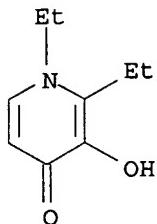
RN 73348-75-1 HCAPLUS
 CN 1,10-Phenanthroline, 2,9-dimethyl-4,7-diphenyl-, disulfo deriv. (9CI) (CA INDEX NAME)



$2 \left[D1 - SO_3H \right]$

RN 115900-75-9 HCAPLUS

CN 4(1H)-Pyridinone, 1,2-diethyl-3-hydroxy- (9CI) (CA INDEX NAME)



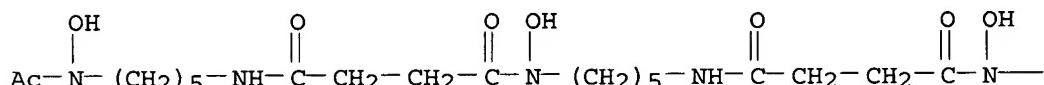
IT 70-51-9, Desferrioxamine

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);
 THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (chelating agents for treatment of **ataxia**
telangiectasia and diseases associated with oxidative stress and
 genomic instability)

RN 70-51-9 HCPLUS

CN Butanediamide, N'-(5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl)-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B

— (CH₂)₅-NH₂

L214 ANSWER 2 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3

ACCESSION NUMBER: 2005:107610 HCPLUS

DOCUMENT NUMBER: 142:422753

TITLE: Pharmacological manipulation of **ataxia-telangiectasia** kinase activity as a treatment for Parkinson's diseaseAUTHOR(S): *Shackelford, Rodney Edwin; Manuszak, Ryan P.; Heard, Steven C.; Link, Charles J.; Wang, Suming*

CORPORATE SOURCE: Department of Pathology, Louisiana State University at Shreveport, Shreveport, LA, 711030-3932, USA

SOURCE: Medical Hypotheses (2005), 64(4), 736-741

CODEN: MEHYDY; ISSN: 0306-9877

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Parkinson's disease (PD) is a major cause of morbidity and mortality among older individuals. Although the causes of Parkinson's disease are multifactorial, considerable evidence indicates that elevated

labile iron in the substantia nigra pars compacta plays an important role in producing oxyradicals which subsequently damage nigro-striatal neurons. Based on this several researchers have suggested that blood-brain barrier crossing iron **chelators** might have clin. efficacy in treating PD. Work demonstrating that iron **chelators** protect nigro-striatal neurons in the N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine and 6-hydroxydopamine-induced rodent PD models supports this hypothesis. Recently, we found that the ATM gene product (mutated in **ataxia-telangiectasia**, A-T), is required for cell survival and genomic stability maintenance following exposure to low labile iron concns. Iron **chelators** (desferal, quercetin, and apoferritin) also increase A-T cell genomic stability and viability, and activate ATM-dependent cellular events in normal cells. Addnl. Atm-deficient mice exhibit a selective loss of dopaminergic nigro-striatal neurons. Based on this, we propose that iron **chelators** protect the substantia nigra pars compacta not only by **chelating** labile iron and reducing oxyradical formation, but also by inducing ATM activity, leading to increased oxidative stress resistance and DNA repair. Support for this hypothesis comes from the recent observation that the iron **chelating** flavonoid quercetin both directly activates ATM and protects neuronal cells from the toxic effects of the N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine. Therefore since; (1) ATM is required for iron toxicity resistance, (2) iron **chelators** such as quercetin, desferal, and apoferritin induce ATM activity and/or ATM-dependent events, and (3), Atm-deficient mice preferentially lose dopaminergic nigro-striatal neurons, we propose that ATM activity has an important function in PD. Furthermore, pharmacol. manipulation of ATM activity via iron **chelation** might have clin. efficacy in PD treatment.

- CC 1-0 (Pharmacology)
 ST review **ataxia telangiectasia** kinase iron
chelation Parkinson disease antiparkinsonian; desferal quercetin
 apoferritin Parkinson disease antiparkinsonian review
 IT Proteins
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (ATM (**ataxia telangiectasia** mutated); pharmacol.
 manipulation of mutated **ataxia telangiectasia**
 kinase activity via iron **chelation** showed efficacy in rodent
 PD models and may increase SNpc neuronal cell survival and slow clin.
 progression of Parkinson's disease)
 IT Ferritins
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (apoferritins; pharmacol. manipulation of mutated **ataxia**
telangiectasia kinase activity via iron **chelator**
 apoferritin may increase SNpc neuronal cell survival and slow clin.
 progression of Parkinson's disease)
 IT Antiparkinsonian agents
 Brain
Chelation
 Parkinson's disease
 (pharmacol. manipulation of mutated **ataxia**
telangiectasia kinase activity via iron **chelation**
 showed efficacy in rodent PD models and may increase SNpc neuronal cell
 survival and slow clin. progression of Parkinson's disease)
 IT Brain
 (substantia nigra, pars compacta; pharmacol. manipulation of mutated
ataxia telangiectasia kinase activity via iron
chelation may increase neuronal cell survival in substantia
 nigra pars compacta and show clin. efficacy in Parkinson's disease)
 IT 7439-89-6, Iron, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (pharmacol. manipulation of mutated **ataxia telangiectasia** kinase activity via iron **chelation**
 showed efficacy in rodent PD models and may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

IT 138-14-7, Desferal

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (pharmacol. manipulation of mutated **ataxia telangiectasia** kinase activity via iron **chelator**
 desferal may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

IT 117-39-5, Quercetin

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (pharmacol. manipulation of mutated **ataxia telangiectasia** kinase activity via iron **chelator**
 quercetin may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

IT 138-14-7, Desferal

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (pharmacol. manipulation of mutated **ataxia telangiectasia** kinase activity via iron **chelator**
 desferal may increase SNpc neuronal cell survival and slow clin. progression of Parkinson's disease)

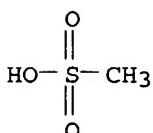
RN 138-14-7 HCPLUS

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy-, monomethanesulfonate (salt) (9CI) (CA INDEX NAME)

CM 1

CRN 75-75-2

CMF C H4 O3 S

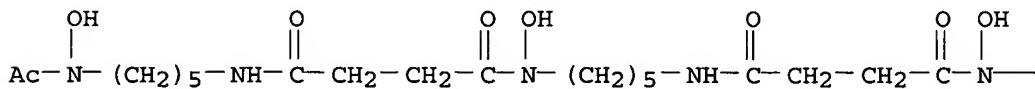


CM 2

CRN 70-51-9

CMF C25 H48 N6 O8

PAGE 1-A

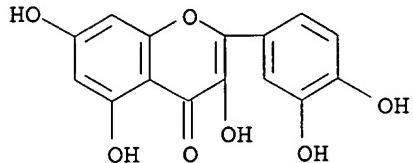


PAGE 1-B

 $-\text{CH}_2\text{CH}_2\text{CH}_2\text{CH}_2\text{NH}_2$

IT 117-39-5, Quercetin
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (pharmacol. manipulation of mutated **ataxia-telangiectasia** kinase activity via iron **chelator**
 quercetin may increase SNpc neuronal cell survival and slow clin.
 progression of Parkinson's disease)

RN 117-39-5 HCPLUS
 CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI)
 (CA INDEX NAME)



REFERENCE COUNT: 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 3 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 4
 ACCESSION NUMBER: 2005:465449 HCPLUS
 DOCUMENT NUMBER: 143:277995
 TITLE: Pharmacologic manipulation of the **ataxia-telangiectasia** mutated gene product as an intervention in age-related disease
Shackelford, Rodney E.
 AUTHOR(S):
 CORPORATE SOURCE: Department of Pathology, Louisiana State University at Shreveport, Shreveport, LA, 711030-3932, USA
 SOURCE: Medical Hypotheses (2005), 65(2), 363-369
 CODEN: MEHYDY; ISSN: 0306-9877
 PUBLISHER: Elsevier Ltd.
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English
 AB A review. **Ataxia-telangiectasia** (A-T) is an autosomal recessive disorder characterized by progressive ataxia, elevated cancer incidence, and premature aging. A-T cells, Atm-deficient mice, and individuals with A-T show increased oxidant sensitivity, genomic instability, altered IGF-1 and p53 signaling, and rapid telomere shortening compared to normal controls. The gene mutated in A-T, ATM, regulates DNA repair, IGF-1 and p53 signaling, age pigment removal, antioxidant capacity, and telomere maintenance - pathways involved in and often attenuated with aging. Interestingly, flavonoids with chemopreventative effects, such as quercetin, genistein, and epigallocatechin gallate activate ATM. Since ATM activates pathways which increase genomic stability, oxidant resistance, and/or telomere stability, and since many diseases of old age (i.e., cancer, cardiovascular and neurodegenerative disease), result from attenuation of these pathways, pharmacol. manipulation of ATM activity via flavonoid intake may prove

useful in slowing the appearance of age-associated disease.

CC 1-0 (Pharmacology)

ST review genetic mutation flavonoid aging **ataxia telangiectasia**

IT Nervous system, disease

(**ataxia telangiectasia**; pharmacol. manipulation of ATM gene activity via flavonoid may be useful in slowing appearance of age-associated disease in human by activating pathways which increase genomic stability, oxidant resistance and telomere stability)

REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 4 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 2004:710500 HCAPLUS

DOCUMENT NUMBER: 141:236524

TITLE: Iron chelators increase the resistance of **Ataxia telangiectasia** cells to oxidative stress

AUTHOR(S): **Shackelford, Rodney E.; Manuszak, Ryan P.; Johnson, Cybele D.; Hellrung, Daniel J.; Link, Charles J.; Wang, Suming**

CORPORATE SOURCE: Iowa Cancer Research Foundation, Urbandale, IA, 50322, USA

SOURCE: DNA Repair (2004), 3(10), 1263-1272

CODEN: DRNEAR; ISSN: 1568-7864

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB **Ataxia telangiectasia** (A-T) is an autosomal recessive disorder characterized by immune dysfunction, genomic instability, chronic oxidative damage, and increased cancer incidence. Previously, desferal was found to increase the resistance of A-T, but not normal cells to exogenous oxidative stress in the colony forming-efficiency assay, suggesting that iron metabolism is dysregulated in A-T. Since desferal both chelates iron and modulates gene expression, the authors tested the effects of apo ferritin and the iron chelating flavonoid quercetin on A-T cell colony-forming ability. The authors demonstrate that apo ferritin and quercetin increase the ability of A-T cells to form colonies. The authors also show that labile iron levels are significantly elevated in Atm-deficient mouse sera compared to syngeneic wild type mice. Our findings support a role for labile iron acting as a Fenton catalyst in A-T, contributing to the chronic oxidative stress seen in this disease. Our findings further suggest that iron chelators might promote the survival of A-T cells and hence, individuals with A-T.

CC 1-12 (Pharmacology)

ST quercetin apo ferritin iron chelator oxidative stress **ataxia telangiectasia**

IT Ferritins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (apo ferritins; iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

IT Nervous system, disease

(**ataxia telangiectasia**; iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

IT Antioxidants

Chelating agents

Oxidative stress, biological

(iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

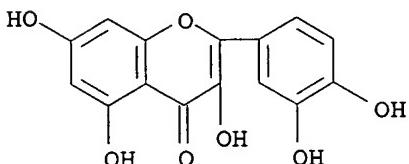
IT Reactive oxygen species
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

IT 7439-89-6, Iron, biological studies 7782-44-7D, Oxygen, reactive species
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

IT 117-39-5, Quercetin
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

IT 117-39-5, Quercetin
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (iron chelators increase resistance of **Ataxia telangiectasia** cells to oxidative stress)

RN 117-39-5 HCPLUS
 CN 4H-1-Benzopyran-4-one, 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxy- (9CI)
 (CA INDEX NAME)



REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 5 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 6
 ACCESSION NUMBER: 2003:985326 HCPLUS
 DOCUMENT NUMBER: 140:175941
 TITLE: Functional expression of ATM gene carried by HSV amplicon vector in vitro and in vivo
 AUTHOR(S): Qi, J.; *Shackelford, R.*; Manuszak, R.; Cheng, D.; Smith, M.; Link, C. J.; Wang, S.
 CORPORATE SOURCE: Human Gene Therapy Research Institute, Stoddard Cancer Research Institute, IA, USA
 SOURCE: Gene Therapy (2004), 11(1), 25-33
 CODEN: GETHEC; ISSN: 0969-7128
 PUBLISHER: Nature Publishing Group
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB **Ataxia-telangiectasia** (AT) is a human autosomal recessive disease with a pleiotropic phenotype characterized by cerebellar degeneration, immunodeficiency, premature aging, cancer predisposition, and radiation sensitivity. The gene mutated in AT, ATM (for AT-mutated), had been cloned and found to have ionizing radiation and oxidative stress-inducible kinase activity. No treatment can stop the progression of the disease. In this study, the complete open-reading frame of ATM cDNA was cloned into a Herpes simplex virus type-1 (HSV-1) amplicon vector (pTO-ATM), and the transduction of cultured AT cells was demonstrated by immunohistochem. and Western blot anal. Functional gene expression was evaluated by cell colony-forming assays following exposure to oxidative stress. The survival of AT cells with ATM gene transduction was about

100% higher compared to nontransduced cells after t-Bu hydroperoxide treatments. Next, the normal ATM gene expression in different regions of the rat brain was studied. Immunohistochem. staining demonstrated weak endogenous ATM protein expression in neurons of the caudate-putamen, with significantly higher levels of expression detected in neurons in other brain regions. Exogenous ATM gene expression from pTO-ATM after viral transduction in the caudate-putamen of the adult rat was examined. At 3 days after injection of the pTO-ATM viral vector, abundant pos. ATM staining of the neurons was found at the injection sites, in comparison to the controls. These data demonstrate that the relatively large ATM cDNA can be transduced and expressed in vitro and in vivo from an HSV amplicon viral vector. These data provide initial evidence that the replacement of the ATM gene into the cells of AT patients might be possible some day.

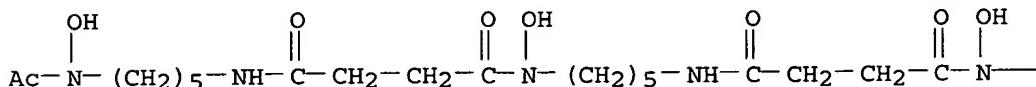
CC 3-2 (Biochemical Genetics)
 Section cross-reference(s): 10, 14
 IT Proteins
 RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
 (ATM (*ataxia telangiectasia* mutated); functional expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)
 IT Nervous system, disease
 (*ataxia telangiectasia*; functional expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)
 IT cDNA
 RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
 (for ATM (*ataxia telangiectasia* mutated); functional expression of ATM cDNA carried by HSV amplicon vector in vitro and in rat brain)
 REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 6 OF 22 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 7
 ACCESSION NUMBER: 2003:705261 HCAPLUS
 DOCUMENT NUMBER: 140:105085
 TITLE: Desferrioxamine treatment increases the genomic stability of *Ataxia-telangiectasia* cells
 AUTHOR(S): *Shackelford, Rodney E.; Manuszak, Ryan P.; Johnson, Cybele D.; Hellrung, Daniel J.; Steele, Timothy A.; Link, Charles J.; Wang, Suming*
 CORPORATE SOURCE: Osteopathic Medical Center, Des Moines University, Des Moines, IA, 50309, USA
 SOURCE: DNA Repair (2003), 2(9), 971-981
 CODEN: DRNEAR; ISSN: 1568-7864
 PUBLISHER: Elsevier Science B.V.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB *Ataxia-telangiectasia* (AT) is an autosomal recessive disorder characterized by genomic instability, chronic oxidative damage, and increased cancer incidence. Compared to normal cells, AT cells exhibit unusual sensitivity to exogenous oxidants, including t-Bu hydroperoxide (t-BOOH). Since ferritin releases labile iron under oxidative stress (which is chronic in AT) and labile iron mediates the toxic effects of t-Bu hydroperoxide, we hypothesized that chelation of intracellular labile iron would increase the genomic stability of AT cells, with and without exogenous oxidative stress. Here we report that desferrioxamine treatment increases the plating efficiency of AT, but not normal cells, in the colony forming-efficiency assay (a

method often used to measure genomic stability). Addnl., desferrioxamine increases AT, but not normal cell resistance, to t-Bu hydroperoxide in this assay. Last, AT cells exhibit increased sensitivity to the toxic effects of FeCl₂ in the colony forming-efficiency assay and fail to demonstrate a FeCl₂-induced G2 checkpoint response when compared to normal cells. Our data indicates that: (1) **chelation** of labile iron increases genomic stability in AT cells, but not normal cells; and (2) AT cells exhibit deficits in their responses to iron toxicity. While preliminary, our findings suggest that AT might be, in part, a disorder of iron metabolism and treatment of individuals with AT with desferrioxamine might have clin. efficacy.

- CC 1-11 (Pharmacology)
 Section cross-reference(s): 14
 ST iron **chelator** desferrioxamine genomic stability **Ataxia telangiectasia**
 IT Nervous system, disease
 (**ataxia telangiectasia**; iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 IT Cell cycle
 (checkpoint, G2; iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 IT **Chelating agents**
 Human
 Oxidative stress, biological
 (iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 IT 75-91-2, tert-Butyl hydroperoxide 7758-94-3, Iron dichloride
 RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
 (iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 IT 50-78-2, Aspirin 70-51-9, Desferrioxamine
 RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 IT 70-51-9, Desferrioxamine
 RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (iron **chelator** desferrioxamine increases genomic stability of **Ataxia telangiectasia** cells: iron metabolism role in AT pathogenesis)
 RN 70-51-9 HCAPLUS
 CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B

— (CH₂)₅—NH₂

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 7 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 2003:695693 HCPLUS
DOCUMENT NUMBER: 140:124579
TITLE: ATM-dependent and -independent gene expression changes in response to oxidative stress, gamma irradiation, and UV irradiation

AUTHOR(S): Heinloth, Alexandra N.; *Shackelford, Rodney E.*; Innes, Cynthia L.; Bennett, Lee; Li, Leping; Amin, Rupesh P.; Sieber, Stella O.; Flores, Kristina G.; Bushel, Pierre R.; Paules, Richard S.

CORPORATE SOURCE: Growth Control and Cancer Group, National Institute of Environmental Health Sciences, Research Triangle Park, NC, 27709, USA

SOURCE: Radiation Research (2003), 160(3), 273-290
CODEN: RAREAE; ISSN: 0033-7587

PUBLISHER: Radiation Research Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB **Ataxia telangiectasia** (AT) is an autosomal recessive disorder characterized by progressive cerebellar degeneration, immunodeficiencies, telangiectasias, sensitivity to ionizing radiation, and high predisposition for malignancies. The **ataxia telangiectasia** mutated (ATM) gene encodes a protein (ATM) with serine/threonine kinase activity. DNA-double strand breaks are known to increase its kinase activity. While cells from individuals with AT are attenuated in their G1-, S- and G2-phase cell cycle checkpoint functions in response to γ irradiation and oxidative stress, their response to UV irradiation appears to be equivalent to that of wild-type cells. In this study,

we investigated changes in gene expression in response to γ irradiation, oxidative stress, and UV irradiation, focusing on the dependence on ATM. Doses for all three treatments were selected that resulted in roughly an equivalent induction of a G1 checkpoint response and inhibition of progression through S phase. To investigate gene expression changes, logarithmically growing wild-type and AT dermal diploid fibroblasts were exposed to either γ radiation (5 Gy), oxidative stress (75 μ M t-butyl-hydroperoxide), or UV radiation (7.5 J/m²), and RNA was harvested 6 h after treatment. Gene expression anal. was performed using the NIEHS Human ToxChip 2.0 with approx. 1900 cDNA clones representing known genes and ESTs. All three treatments resulted in distinct patterns of gene expression changes, as shown previously. ATM-dependent and ATM-independent components were detected within these patterns, as were novel indications of involvement of ATM in regulation of transcription factors such as SP1, AP1 and MTF1.

CC 8-7 (Radiation Biochemistry)

IT Nervous system, disease

(ataxia telangiectasia; ATM-dependent and -independent gene expression changes in response to oxidative stress, γ -irradiation, and UV)

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 8 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 9
 ACCESSION NUMBER: 2001:455746 HCPLUS
 DOCUMENT NUMBER: 135:193892
 TITLE: The *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts
 AUTHOR(S): *Shackelford, Rodney E.; Innes, Cynthia L.; Sieber, Stella O.; Heinloth, Alexandra N.; Leadon, Steven A.; Paules, Richard S.*
 CORPORATE SOURCE: Growth Control and Cancer Group, NIEHS, National Institutes of Health, Research Triangle Park, NC, 27709, USA
 SOURCE: Journal of Biological Chemistry (2001), 276(24), 21951-21959
 CODEN: JBCHA3; ISSN: 0021-9258
 PUBLISHER: American Society for Biochemistry and Molecular Biology
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB *Ataxia telangiectasia* (AT) is an autosomal recessive disorder characterized by neuronal degeneration accompanied by *ataxia, telangiectasias*, acute cancer pre-disposition, and sensitivity to ionizing radiation (IR). Cells from individuals with AT show unusual sensitivity to IR, severely attenuated cell cycle checkpoint functions, and poor p53 induction in response to IR compared with normal human fibroblasts (NHFs). The gene mutated in AT (ATM) has been cloned, and its product, pATM, has IR-inducible kinase activity. The AT phenotype has been suggested to be a consequence, at least in part, of an inability to respond appropriately to oxidative damage. To test this hypothesis, we examined the ability of NHFs and AT dermal fibroblasts to respond to t-Bu hydroperoxide and IR treatment. AT fibroblasts exhibit, in comparison to NHFs, increased sensitivity to the toxicity of t-Bu hydroperoxide, as measured by colony-forming efficiency assays. Unlike NHFs, AT fibroblasts fail to show G1 and G2 phase checkpoint functions or to induce p53 in response to t-Bu hydroperoxide. Treatment of NHFs with t-Bu hydroperoxide activates pATM-associated kinase activity. Our results indicate that pATM is involved in responding to certain aspects of oxidative damage and in signaling this information to downstream effectors of the cell cycle checkpoint functions. Our data further suggest that some of the pathologies seen in AT could arise as a consequence of an inability to respond normally to oxidative damage.

CC 14-10 (Mammalian Pathological Biochemistry)
 ST *Ataxia telangiectasia* gene oxidative stress cell cycle fibroblast
 IT Gene, animal
 RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
 (AT; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)
 IT Fibroblast
 Oxidative stress, biological
 Phenotypes
 Signal transduction, biological
 (*Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)
 IT p53 (protein)

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(*Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Interphase (cell cycle)

(G1-phase; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Interphase (cell cycle)

(G2-phase; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Neoplasm

(acute cancer pre-disposition; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Nervous system

(*ataxia telangiectasia*; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Nerve

(degeneration; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT Ionizing radiation

(sensitivity to; *Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

IT 182970-53-2, gene ATM protein

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(*Ataxia telangiectasia* gene product is required for oxidative stress-induced G1 and G2 checkpoint function in human fibroblasts)

REFERENCE COUNT:

50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 9 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 10

ACCESSION NUMBER: 2001:512887 HCPLUS

DOCUMENT NUMBER: 135:267656

TITLE: Caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated (Atm) gene function

AUTHOR(S): Morita, Y.; Maravei, D. V.; Bergeron, L.; Wang, S.; Perez, G. I.; Tsutsumi, O.; Taketani, Y.; Asano, M.; Horai, R.; Korsmeyer, S. J.; Iwakura, Y.; Yuan, J.; Tilly, J. L.

CORPORATE SOURCE: Vincent Center for Reproductive Biology, Department of Obstetrics and Gynecology, Massachusetts General Hospital/Harvard Medical School, Boston, MA, 02114, USA

SOURCE: Cell Death and Differentiation (2001), 8(6), 614-620
CODEN: CDDIEK; ISSN: 1350-9047

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It is well established that programmed cell death claims up to two-thirds

of the oocytes produced during gametogenesis in the developing fetal ovaries. However, the mechanisms underlying prenatal germ cell loss in females remain poorly understood. Herein the authors report that caspase-11 null female mice are born with a reduced number of oocyte-containing primordial follicles. This phenotype is likely due to failed cytokine processing known to occur in caspase-11 mutants since neonatal female mice lacking both interleukin (IL)-1 α and IL-1 β also exhibit a reduced endowment of primordial follicles. In addition, germ cell death in wild-type fetal ovaries cultured ex vivo is suppressed by either cytokine, likely via ligand activation of type 1 IL-1 receptors expressed in fetal germ cells. Normal oocyte endowment can be restored in caspase-11 null female mice by simultaneous inactivation of the gene encoding the cell death executioner enzyme, caspase-2. However, caspase-2 deficiency cannot overcome gametogenic failure resulting from meiotic recombination defects in *ataxia telangiectasia*-mutated (Atm) null female mice. Thus, genetically distinct mechanisms exist for developmental deletion of oocytes via programmed cell death, one of which probably functions as a meiotic quality-control checkpoint that cannot be overridden.

- CC 2-10 (Mammalian Hormones)
 Section cross-reference(s): 13
- ST IL1 caspase ovary germ cell apoptosis **ataxia telangiectasia**
- IT Gene, animal
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (Bax; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Nervous system
 (*ataxia telangiectasia*; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Apoptosis
 Gamete and Germ cell
 Meiosis
 (caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Interleukin 1 α
 Interleukin 1 β
 RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Ovary
 (follicle; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Egg
 (oocyte; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of *ataxia telangiectasia*-mutated gene function)
- IT Interleukin 1 receptors

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(type I; caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of **ataxia telangiectasia**-mutated gene function)

IT 182372-14-1, caspase 2 216503-96-7, caspase 11

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(caspase-2 deficiency prevents programmed germ cell death resulting from cytokine insufficiency but not meiotic defects caused by loss of **ataxia telangiectasia**-mutated gene function)

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 10 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 11

ACCESSION NUMBER: 1999:725087 HCPLUS

DOCUMENT NUMBER: 132:62619

TITLE: Lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of nuclear factor- κ B (NF- κ B) in human diploid fibroblasts

AUTHOR(S): Ashburner, Brian P.; **Shackelford, Rodney E.**; Baldwin, Albert S., Jr.; Paules, Richard S.

CORPORATE SOURCE: Lineberger Comprehensive Cancer Center, University of North Carolina School of Medicine, Chapel Hill, NC, 27599, USA

SOURCE: Cancer Research (1999), 59(21), 5456-5460

CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: AACR Subscription Office

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It has been suggested that the cellular response to exposure to ionizing radiation involves activation of the transcription factor nuclear factor- κ B (NF- κ B) and that this response is defective in cells from individuals with **ataxia telangiectasia** (AT). In one study, it was found that SV40 large T-transformed cells derived from a patient null for the AT mutated (ATM) gene exhibited constitutive activation of NF- κ B and that in those cells, inhibition of NF- κ B by expression of a modified form of I κ B α led to correction of the radiosensitivity associated with the AT phenotype. From those data, it was suggested that NF- κ B played a role in the AT phenotype. We show here that normal diploid cells derived from AT patients do not exhibit constitutive activation of NF- κ B. Furthermore, we provide data that the transformation process associated with SV40 large T antigen expression in AT-/ cells leads to aberrant cellular responses. Our studies highlight the importance of using diploid, nontransformed AT-/ cells for in vitro studies relevant to the AT phenotype whenever possible.

CC 14-14 (Mammalian Pathological Biochemistry)

Section cross-reference(s): 3, 8

ST **ataxia telangiectasia** gene ATM NF κ B fibroblast

IT Gene, animal

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (ATM; lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Phosphoproteins

RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); BIOL (Biological study)
 (I κ B- α (inhibitor of RNA formation factor NF- κ B,
 α); lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Transcription factors
 RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (NF- κ B (nuclear factor κ B); lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Nervous system
 (**ataxia telangiectasia**; lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Transformation, neoplastic
 (fibroblast; lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Fibroblast
 Ionizing radiation
 (lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

IT Fibroblast
 (transformation; lack of involvement of **ataxia telangiectasia** mutated (ATM) in regulation of NF- κ B in human diploid fibroblasts in relation to radiosensitivity)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 11 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 12
 ACCESSION NUMBER: 1999:221513 HCPLUS
 DOCUMENT NUMBER: 131:54809
 TITLE: Cell cycle control, checkpoint mechanisms, and genotoxic stress
 AUTHOR(S): *Shackelford, Rodney E.; Kaufmann, William K.; Paules, Richard S.*
 CORPORATE SOURCE: Growth Control and Cancer Group, National Institute of Environmental Health Sciences, Research Triangle Park, NC, 27709, USA
 SOURCE: Environmental Health Perspectives Supplements (1999), 107(1), 5-24
 CODEN: EHPSEO; ISSN: 1078-0475
 PUBLISHER: National Institute of Environmental Health Sciences
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English
 AB A review with 490 refs. The ability of cells to maintain genomic integrity is vital for cell survival and proliferation. Lack of fidelity in DNA replication and maintenance can result in deleterious mutations leading to cell death or, in multicellular organisms, cancer. The purpose of this review is to discuss the known signal transduction pathways that regulate cell cycle progression and the mechanisms cells employ to insure DNA stability in the face of genotoxic stress. In particular, we focus on mammalian cell cycle checkpoint functions, their role in maintaining DNA stability during the cell cycle following exposure to genotoxic agents, and the gene products that act in checkpoint function signal transduction

cascades. Key transitions in the cell cycle are regulated by the activities of various protein kinase complexes composed of cyclin and cyclin-dependent kinase (Cdk) mols. Surveillance control mechanisms that check to ensure proper completion of early events and cellular integrity before initiation of subsequent events in cell cycle progression are referred to as cell cycle checkpoints and can generate a transient delay that provides the cell more time to repair damage before progressing to the next phase of the cycle. A variety of cellular responses are elicited that function in checkpoint signaling to inhibit cyclin/Cdk activities. These responses include the p53-dependent and p53-independent induction of Cdk inhibitors and the p53-independent inhibitory phosphorylation of Cdk mols. themselves. Eliciting proper G1, S, and G2 checkpoint responses to double-strand DNA breaks requires the function of the **Ataxia telangiectasia** mutated gene product. Several human heritable cancer-prone syndromes known to alter DNA stability have been found to have defects in checkpoint surveillance pathways. Exposures to several common sources of genotoxic stress, including oxidative stress, ionizing radiation, UV radiation, and the genotoxic compound benzo[a]pyrene, elicit cell cycle checkpoint responses that show both similarities and differences in their mol. signaling.

CC 4-0 (Toxicology)

Section cross-reference(s): 8, 14

REFERENCE COUNT: 490 THERE ARE 490 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L214 ANSWER 12 OF 22 HCPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:38566 HCPLUS

DOCUMENT NUMBER: 144:485602

TITLE: Increased transferrin receptor expression following 11q23 deletion as a mechanism of malignant progression in chronic lymphocytic leukemia

AUTHOR(S): **Shackelford, Rodney E.**; Bhalodia, Ami R.; Cotelingam, James D.; Veillon, Diana M.;

Lowery-Nordberg, Mary

CORPORATE SOURCE: Department of Pathology, Louisiana State University at Shreveport, Shreveport, LA, 711030-3932, USA

SOURCE: Medical Hypotheses (2005), Volume Date 2006, 66(3), 509-512

CODEN: MEHYDY; ISSN: 0306-9877

Elsevier Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Chronic lymphocytic leukemia (CLL) is a common adult leukemia characterized by the accumulation of mature neoplastic B-lymphocytes. Typically, CLL follows an indolent course, with most patients surviving for many years. However, 10-20% of CLL patients carry 11q23 chromosomal deletions and often exhibit a more severe disease course, with earlier onset of symptoms, shortened lymphocyte doubling time, poor response to therapy, and shortened survival. The mol. basis for 11q23 deletions resulting in a poor prognosis is currently poorly understood. The tumor suppressor gene, **ataxia-telangiectasia** mutated (ATM, 11q22.3-23.1), is considered a likely candidate gene whose loss could result in the poor prognosis associated with 11q23 deletion and is mutated in a significant percentage of CLL cases. Recently, recombinant ATM expression in ATM-deficient cells was found to decrease transferrin receptor (TfR) expression, suggesting that deletion of the chromosomal region carrying ATM results in increased TfR expression. TfR imports iron into cells, an event necessary for DNA synthesis and cell growth. Addnl., rapidly growing malignant cells, including lymphomas and CLL, often

express high TfR levels. Based on this, we propose that one mol. mechanism by which 11q23 deletions confer a poor prognosis in CLL is via increased TfR expression secondary to ATM loss, resulting in the increased cellular iron import, and hence increased capacity for malignant growth. Our hypothesis may also partially explain why gallium, an atomically iron-like toxic metal that binds to transferrin and the TfR is incorporated into cells and was previously demonstrated to have anti-tumor activity in patients with lymphomas refractory to other chemotherapeutic treatments.

CC 14-0 (Mammalian Pathological Biochemistry)

Section cross-reference(s): 3

IT Proteins

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)

(ATM (*ataxia telangiectasia* mutated); chromosomal

11q23 deletion may confer poor prognosis in CLL patient via increased TfR expression secondary to tumor suppressor gene ATM loss resulting in cellular iron import hence increased capacity for malignant growth)

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS, MEDLINE, BIOSIS' - CONTINUE? (Y)/N:y

L214 ANSWER 13 OF 22 MEDLINE on STN DUPLICATE 1
 ACCESSION NUMBER: 2006016513 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 16326028
 TITLE: Increased transferrin receptor expression following 11q23 deletion as a mechanism of malignant progression in chronic lymphocytic leukemia.
 AUTHOR: *Shackelford Rodney E; Bhalodia Ami R; Cotelingam James D; Veillon Diana M; Lowery-Nordberg Mary*
 CORPORATE SOURCE: Louisiana State University at Shreveport, Department of Pathology, 1501 Kings Hwy, P.O. Box 33932, Shreveport, LA 711030-3932, USA.. RdnyShac@aol.com
 SOURCE: Medical hypotheses, (2006) Vol. 66, No. 3, pp. 509-12.
 Electronic Publication: 2005-12-02.
 Journal code: 7505668. ISSN: 0306-9877.
 PUB. COUNTRY: Scotland: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200606
 ENTRY DATE: Entered STN: 11 Jan 2006
 Last Updated on STN: 23 Jun 2006
 Entered Medline: 22 Jun 2006

ABSTRACT:

Chronic lymphocytic leukemia (CLL) is a common adult leukemia characterized by the accumulation of mature neoplastic B-lymphocytes. Typically, CLL follows an indolent course, with most patients surviving for many years. However, 10-20% of CLL patients carry 11q23 chromosomal deletions and often exhibit a more severe disease course, with earlier onset of symptoms, shortened lymphocyte doubling time, poor response to therapy, and shortened survival. The molecular basis for 11q23 deletions resulting in a poor prognosis is currently poorly understood. The tumor suppressor gene, *ataxia-telangiectasia* mutated (ATM, 11q22.3-23.1), is considered a likely candidate gene whose loss could result in the poor prognosis associated with 11q23 deletion and is

mutated in a significant percentage of CLL cases.. Recently, recombinant ATM expression in ATM-deficient cells was found to decrease transferrin receptor (TfR) expression, suggesting that deletion of the chromosomal region carrying ATM results in increased TfR expression. TfR imports iron into cells, an event necessary for DNA synthesis and cell growth. Additionally, rapidly growing malignant cells, including lymphomas and CLL, often express high TfR levels. Based on this, we propose that one molecular mechanism by which 11q23 deletions confer a poor prognosis in CLL is via increased TfR expression secondary to ATM loss, resulting in the increased cellular iron import, and hence increased capacity for malignant growth. Our hypothesis may also partially explain why gallium, an atomically iron-like toxic metal that binds to transferrin and the TfR is incorporated into cells and was previously demonstrated to have anti-tumor activity in patients with lymphomas refractory to other chemotherapeutic treatments.

CONTROLLED TERM: B-Lymphocytes: PA, pathology
 Cell Cycle Proteins: GE, genetics
 Chromosome Deletion
 *Chromosomes, Human, Pair 11
 DNA-Binding Proteins: GE, genetics
 Disease Progression
 *Gene Deletion
 Humans
 *Leukemia, Lymphocytic, Chronic: GE, genetics
 Lymphocytes: ME, metabolism
 Prognosis
 Protein-Serine-Threonine Kinases: GE, genetics
 *Receptors, Transferrin: BI, biosynthesis
 Receptors, Transferrin: GE, genetics
 Recombinant Proteins: ME, metabolism
 Tumor Suppressor Proteins: GE, genetics
 0 (Cell Cycle Proteins); 0 (DNA-Binding Proteins); 0 (Receptors, Transferrin); 0 (Recombinant Proteins); 0 (Tumor Suppressor Proteins); EC 2.7.1.37 (Protein-Serine-Threonine Kinases); EC 2.7.1.37 (ataxia telangiectasia mutated protein)

CHEMICAL NAME:

L214 ANSWER 14 OF 22 MEDLINE on STN
 ACCESSION NUMBER: 2000144083 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10677503
 TITLE: The catalytic subunit of DNA-dependent protein kinase selectively regulates p53-dependent apoptosis but not cell-cycle arrest.
 AUTHOR: Wang S; Guo M; Ouyang H; Li X; Cordon-Cardo C; Kurimasa A; Chen D J; Fuks Z; Ling C C; Li G C
 CORPORATE SOURCE: Department of Medical Physics, Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York, NY 10021; and Los Alamos National Laboratory, Los Alamos, NM 87545, USA.
 CONTRACT NUMBER: CA-31397 (NCI)
 CA-56909 (NCI)
 CA-78497 (NCI)
 +
 SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2000 Feb 15) Vol. 97, No. 4, pp. 1584-8.
 Journal code: 7505876. ISSN: 0027-8424.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200003

ENTRY DATE: Entered STN: 30 Mar 2000
 Last Updated on STN: 20 Apr 2002
 Entered Medline: 23 Mar 2000

ABSTRACT:

DNA damage induced by ionizing radiation (IR) activates p53, leading to the regulation of downstream pathways that control cell-cycle progression and apoptosis. However, the mechanisms for the IR-induced p53 activation and the differential activation of pathways downstream of p53 are unclear. Here we provide evidence that the catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) serves as an upstream effector for p53 activation in response to IR, linking DNA damage to apoptosis. DNA-PKcs knockout (DNA-PKcs-/-) mice were exposed to whole-body IR, and the cell-cycle and apoptotic responses were examined in their thymuses. Our data show that IR induction of apoptosis and Bax expression, both mediated via p53, was significantly suppressed in the thymocytes of DNA-PKcs-/- mice. In contrast, IR-induced cell-cycle arrest and p21 expression were normal. Thus, DNA-PKcs deficiency selectively disrupts p53-dependent apoptosis but not cell-cycle arrest. We also confirmed previous findings that p21 induction was attenuated and cell-cycle arrest was defective in the thymocytes of whole body-irradiated Atm-/- mice, but the apoptotic response was unperturbed. Taken together, our results support a model in which the upstream effectors DNA-PKcs and Atm selectively activate p53 to differentially regulate cell-cycle and apoptotic responses. Whereas Atm selects for cell-cycle arrest but not apoptosis, DNA-PKcs selects for apoptosis but not cell-cycle arrest.

CONTROLLED TERM: Animals

- *Apoptosis: GE, genetics
- Apoptosis: RE, radiation effects
- Ataxia Telangiectasia: GE, genetics**
- *Cell Cycle: GE, genetics
- Cell Cycle: RE, radiation effects
- DNA Repair: RE, radiation effects
- DNA-Activated Protein Kinase
- *DNA-Binding Proteins
- Flow Cytometry
- In Situ Nick-End Labeling
- Mice
- Mice, Knockout
- *Protein-Serine-Threonine Kinases: GE, genetics
- Protein-Serine-Threonine Kinases: ME, metabolism
- Proto-Oncogene Proteins: ME, metabolism
- *Proto-Oncogene Proteins c-bcl-2
- Research Support, U.S. Gov't, Non-P.H.S.
- Research Support, U.S. Gov't, P.H.S.
- Thymus Gland: PA, pathology
- Thymus Gland: RE, radiation effects
- *Tumor Suppressor Protein p53: ME, metabolism
- Whole-Body Irradiation
- bcl-2-Associated X Protein

CHEMICAL NAME: 0 (Bax protein, mouse); 0 (DNA-Binding Proteins); 0 (Proto-Oncogene Proteins); 0 (Proto-Oncogene Proteins c-bcl-2); 0 (Tumor Suppressor Protein p53); 0 (bcl-2-Associated X Protein); EC 2.7.1.37 (DNA-Activated Protein Kinase); EC 2.7.1.37 (Protein-Serine-Threonine Kinases)

L214 ANSWER 15 OF 22 MEDLINE on STN

ACCESSION NUMBER: 1998347138 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9682216

TITLE: A model for ATM heterozygote identification in a large population: four founder-effect ATM mutations identify most

of Costa Rican patients with **ataxia telangiectasia**.

AUTHOR: Telatar M; Wang S; Castellvi-Bel S; Tai L Q;
Sheikhavandi S; Regueiro J R; Porras O; Gatti R A
CORPORATE SOURCE: Department of Pathology, University of California at Los Angeles School of Medicine 90095, USA.
SOURCE: Molecular genetics and metabolism, (1998 May) Vol. 64, No. 1, pp. 36-43.
Journal code: 9805456. ISSN: 1096-7192.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 20 Oct 1998
Last Updated on STN: 20 Oct 1998
Entered Medline: 6 Oct 1998

ABSTRACT:

Ataxia telangiectasia (A-T) is an autosomal recessive disorder with a broad range of clinical manifestations and a frequency of 1:40,000-100,000 live births. Epidemiological studies have suggested that A-T heterozygotes are at an elevated risk of breast cancer. ATM mutations occur worldwide over the entire ATM gene, making it difficult to identify heterozygotes in large populations. However, some founder-effect mutations are specific for certain populations. Here, we present four mutations in Costa Rican A-T patients that accounted for 86-93% of 41 patients studied in two batches. We have developed assays for rapid detection of these four mutations which can be used diagnostically. They will also enable the Costa Rican population to be used as a model for analyzing the role of ATM heterozygosity in cancer development and other disorders.

CONTROLLED TERM: **Ataxia Telangiectasia: DI, diagnosis**
***Ataxia Telangiectasia: GE, genetics**
Codon, Terminator
Costa Rica
Exons: GE, genetics
*Founder Effect
Genes, Recessive
*Genetic Screening: MT, methods
*Haplotypes
*Heterozygote Detection
Humans
Point Mutation
Restriction Mapping
Sequence Deletion

CHEMICAL NAME: O (Codon, Terminator)

L214 ANSWER 16 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN

ACCESSION NUMBER: 2006:170137 BIOSIS
DOCUMENT NUMBER: PREV200600171437
TITLE: **Ataxia-telangiectasia mutated gene product activity increases resistance to Aspergillus fumigatus gliotoxin toxicity.**
AUTHOR(S): **Shackelford, R. E. [Reprint Author]; Fu, Y.; Abdelbaqi, M.; Lowery-Nordberg, M.; Chen, A.**
CORPORATE SOURCE: Louisiana State Univ, Shreveport, LA 71105 USA
SOURCE: Modern Pathology, (JAN 2006) Vol. 19, No. Suppl. 1, pp. 257A-258A.
Meeting Info.: 95th Annual Meeting of the United-States-and-Canadian-Academy-of-Pathology. Atlanta,

DOCUMENT TYPE: GA, USA. February 11 -17, 2006. US & Canadian Acad Pathol.
 ISSN: 0893-3952.
 Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 9 Mar 2006
 Last Updated on STN: 9 Mar 2006
 CONCEPT CODE: General biology - Symposia, transactions and proceedings
 00520
 Genetics - Plant 03504
 Biochemistry studies - General 10060
 Toxicology - General and methods 22501
 Plant physiology - Chemical constituents 51522
 INDEX TERMS: Major Concepts
 Toxicology; Biochemistry and Molecular Biophysics
 INDEX TERMS: Chemicals & Biochemicals
 ATM; gliotoxin
 ORGANISM: Classifier
 Fungi Imperfecti or Deuteromycetes 15500
 Super Taxa
 Fungi; Plantae
 Organism Name
 Aspergillus fumigatus (species)
 Taxa Notes
 Fungi, Microorganisms, Nonvascular Plants, Plants
 67-99-2 (gliotoxin)
 GENE NAME: Aspergillus fumigatus ATM gene [Aspergillus fumigatus
 ataxia telangiectasia mutated gene]
 (Fungi Imperfecti or Deuteromycetes)

L214 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
 STN
 ACCESSION NUMBER: 2005:414873 BIOSIS
 DOCUMENT NUMBER: PREV200510210084
 TITLE: The **ataxia-telangiectasia** gene product
 is required for genomic stability following labile ferric
 iron exposure.
 AUTHOR(S): **Shackelford, R. E.** [Reprint Author]; Manuszak, R.
 P.; Wang, S.; Lowery-Norberg, M.; Chen, A.
 CORPORATE SOURCE: Louisiana State Univ, Shreveport, LA 71105 USA
 SOURCE: Modern Pathology, (JAN 2005) Vol. 18, No. Suppl. 1, pp.
 301A.
 Meeting Info.: 94th Annual Meeting of the
 United-States-and-Canadian-Academy-of-Pathology. San
 Antonio, TX, USA. February 26 -March 04, 2005. US Canadian
 Acad Pathol.
 ISSN: 0893-3952.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 19 Oct 2005
 Last Updated on STN: 19 Oct 2005
 CONCEPT CODE: General biology - Symposia, transactions and proceedings
 00520
 Genetics - General 03502
 Genetics - Human 03508
 Biochemistry studies - General 10060
 Cardiovascular system - Blood vessel pathology 14508
 Nervous system - Pathology 20506
 Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS: Major Concepts
 Molecular Genetics (Biochemistry and Molecular Biophysics)

INDEX TERMS: Diseases
ataxia-telangiectasia: vascular disease, genetic disease, immune system disease, nervous system disease
Ataxia Telangiectasia (MeSH)

INDEX TERMS: Chemicals & Biochemicals
 ferric iron; **desferal**: chelating agent; apoferritin: chelating agent; **quercetin**: chelating agent; **epigallocatechin-3 gallate**: chelating agent

INDEX TERMS: Miscellaneous Descriptors
 cell viability; genomic stability; ferric iron exposure

REGISTRY NUMBER: 20074-52-6 (ferric iron)
 138-14-7 (**desferal**)
 117-39-5 (**quercetin**)
 989-51-5 (**epigallocatechin-3 gallate**)

GENE NAME: human ATM gene gene (Hominidae)

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ACCESSION NUMBER: 2005:523626 BIOSIS

DOCUMENT NUMBER: PREV200510313589

TITLE: A MAP4K4-TRF2 cycle amplifies apoptotic signals in mouse myocardium.

AUTHOR(S): Xie, Min [Reprint Author]; Wang, Sam C.; Zhang, Dou; Prahash, Arun J.; Oh, Hidemasa; Sano, Motoaki; Wang, Xiaozhen; Pocius, Jennifer S.; Taffet, George E.; Michael, Lloyd H.; Tan, Tse-Hua; Entman, Mark L.; Schneider, Michael D.

CORPORATE SOURCE: Baylor Coll Med, Houston, TX 77030 USA

SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 1.

Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.

CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 1 Dec 2005

Last Updated on STN: 1 Dec 2005

ABSTRACT: Background: Loss of TRF2 (Telomeric Repeat-binding Factor-2) causes apoptosis in cardiomyocytes and other cell types, but how it actuates apoptosis is unknown. Here, we studied the HPK/GCK-like Kinase (HGK, MAP4K4) and its potential relation to telomere dysfunction in cardiomyocytes. Results: (1) In mouse myocardium, each of four biological signals for cardiomyocyte apoptosis induced the kinase activity of HGK: biomechanical stress, ischemia/reperfusion injury, and gain-of-function mutations for TNF alpha or Gq. (2) Analogous results were seen in cultured cardiomyocytes, using oxidative stress, ceramide, doxorubicin, and Gq. (3) HGK-induced apoptosis occurred via TAK1 (MAP3K7), JNK, and dissipation of mitochondrial membrane potential. (4) Interrupting normal TRF2 function in cardiomyocytes with dnTRF2 elicited phosphorylation of p53 and histone H2AX, targets for the *ataxia-telangiectasia* mutated (ATM) DNA damage signaling pathway. (5) Evidence placing TRF2 upstream of the apoptotic HGK-TAK1-JNK signaling module includes: dnTRF2 or knockdown of TRF2

by antisense oligos activated HGK in cardiomyocytes and/or mice; TRF2 reduced basal HGK activity, and apoptosis due to dnTRF2 was largely blocked by dnTAK1 or dnJNK1. (6) Activation of the HGK-TAK1-JNK pathway, in turn, markedly reduced TRF2 levels. (7) Conversely, dominant-negative mutations of the kinases protected TRF2 loss induced by ceramide, a potent activator of HGK. (8) To test the predicted function of TRF2 in vivo, we created alpha MHC driven gain-of-function and dominant-inhibitory (cm mutations. As anticipated, the dnTRF2 transgene caused HGK activation, myocardial apoptosis, dysfunction, and premature mortality in mice. (9) Conversely, wild-type TRF2 conferred protection against the apoptotic cardiomyopathy provoked by doxorubicin.

Conclusions: Activation of the MAP4K, HGK, is a highly generalizable response to pro-apoptotic stress signals in cultured cardiomyocytes and the intact heart. Because loss of TRF2 activates the HGK pathway and activation of the HGK pathway induces TRF2 loss, our findings suggest that loss of TRF2 is reciprocally coupled to activity of the HGK death pathway, as a positive feedback loop that amplifies apoptotic signals.

CONCEPT CODE:

- General biology - Symposia, transactions and proceedings 00520
- Cytology - Animal 02506
- Biochemistry studies - General 10060
- Biochemistry studies - Proteins, peptides and amino acids 10064
- Biochemistry studies - Lipids 10066
- Cardiovascular system - Physiology and biochemistry 14504
- Endocrine - General 17002
- Muscle - Physiology and biochemistry 17504

INDEX TERMS:

- Major Concepts
- Cardiovascular System (Transport and Circulation)

INDEX TERMS:

- Parts, Structures, & Systems of Organisms
- heart: circulatory system; cardiomyocyte: muscular system, circulatory system; myocardium: muscular system, circulatory system

INDEX TERMS:

- Chemicals & Biochemicals
- TNF-alpha [tumor necrosis factor-alpha]; p53; ceramide; doxorubicin; H2AX; telomeric repeat-binding factor-2 [TRF2]; HPK-GCK-like kinase [MAP4K4]

INDEX TERMS:

- Miscellaneous Descriptors
- oxidative stress; biomechanical stress; premature mortality

ORGANISM:

- Classifier
- Muridae 86375
- Super Taxa
- Rodentia; Mammalia; Vertebrata; Chordata; Animalia
- Organism Name
- mouse (common)
- Taxa Notes
- Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER: 104404-17-3 (ceramide)
23214-92-8 (doxorubicin)

L214 ANSWER 19 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:476393 BIOSIS

DOCUMENT NUMBER: PREV200300476393

TITLE: Functional expression of ATM gene carried by HSV amplicon vector in vitro and in vivo.

AUTHOR(S): Qi, Jianguo [Reprint Author]; Manuszak, Ryan [Reprint Author]; **Shackelford, Rodney** [Reprint Author]; Cheng, Dong [Reprint Author]; Smith, Michael [Reprint

CORPORATE SOURCE:
SOURCE:

Author]; Link, Charles J. Jr. [Reprint Author]; Wang, Suming [Reprint Author]
Stoddard Cancer Research Institute, Des Moines, IA, USA
Proceedings of the American Association for Cancer Research Annual Meeting, (July 2003) Vol. 44, pp. 1097. print.
Meeting Info.: 94th Annual Meeting of the American Association for Cancer Research. Washington, DC, USA. July 11-14, 2003.
ISSN: 0197-016X.

DOCUMENT TYPE:

Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 15 Oct 2003

CONCEPT CODE:

General biology - Symposia, transactions and proceedings 00520

Cytology - Animal 02506

Genetics - General 03502

Genetics - Animal 03506

Biochemistry studies - Proteins, peptides and amino acids 10064

Cardiovascular system - Physiology and biochemistry 14504

Cardiovascular system - Blood vessel pathology 14508

Nervous system - Physiology and biochemistry 20504

Nervous system - Pathology 20506

Genetics of bacteria and viruses 31500

Virology - General and methods 33502

Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS:

Major Concepts

Cardiovascular System (Transport and Circulation);
Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination)

INDEX TERMS:

Parts, Structures, & Systems of Organisms

caudate: nervous system; neuron: nervous system;
putamen: nervous system

INDEX TERMS:

Diseases

Ataxia-telangiectasia: genetic disease, immune system disease, nervous system disease, vascular disease, genetics

Ataxia Telangiectasia (MeSH)

INDEX TERMS:

Chemicals & Biochemicals

ataxia-telangiectasia cDNA [
ataxia-telangiectasia complementary DNA]

INDEX TERMS:

Methods & Equipment

Western blot: genetic techniques, laboratory techniques; gamma-ray irradiation: clinical techniques, therapeutic and prophylactic techniques; immunohistochemistry: immunologic techniques, laboratory techniques

INDEX TERMS:

Miscellaneous Descriptors

cell survival rate; oxidative stress

ORGANISM:

Classifier

Herpesviridae 03115

Super Taxa

dsDNA Viruses; Viruses; Microorganisms

Organism Name

Herpes simplex virus type-1 (common) [Human herpesvirus 1 (species)]: gene vector

Taxa Notes

Double-Stranded DNA Viruses, Microorganisms, Viruses

ORGANISM: Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 rat (common): adult
 Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates,
 Nonhuman Mammals, Rodents, Vertebrates
 GENE NAME: rat ATM gene (Muridae): expression, open reading frame

L214 ANSWER 20 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
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ACCESSION NUMBER: 2002:395524 BIOSIS
 DOCUMENT NUMBER: PREV200200395524
 TITLE: ATM-dependent gene expression changes after different forms
 of DNA damage.
 AUTHOR(S): Heinloth, Alexandra N. [Reprint author]; *Shackelford,*
Rodney E. [Reprint author]; Innes, Cynthia L. [Reprint
 author]; Amin, Rupesh P. [Reprint author]; Sieber, Stella
 G. [Reprint author]; Flores, Kristina G. [Reprint author];
 Bennett, Lee [Reprint author]; Bushel, Pierre R. [Reprint
 author]; Paules, Richard S. [Reprint author]

CORPORATE SOURCE: National Institute of Environmental Health Sciences,
 Research Triangle Park, NC, USA
 SOURCE: Proceedings of the American Association for Cancer Research
 Annual Meeting, (March, 2002) Vol. 43, pp. 626. print.
 Meeting Info.: 93rd Annual Meeting of the American
 Association for Cancer Research. San Francisco, California,
 USA. April 06-10, 2002.
 ISSN: 0197-016X.

DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LANGUAGE: English
 ENTRY DATE: Entered STN: 24 Jul 2002
 Last Updated on STN: 29 Aug 2002

CONCEPT CODE: General biology - Symposia, transactions and proceedings
 00520
 Genetics - General 03502
 Genetics - Human 03508
 Biochemistry studies - Nucleic acids, purines and
 pyrimidines 10062
 Cardiovascular system - Physiology and biochemistry 14504
 Cardiovascular system - Blood vessel pathology 14508
 Integumentary system - Physiology and biochemistry 18504
 Nervous system - Physiology and biochemistry 20504
 Nervous system - Pathology 20506
 Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS: Major Concepts
 Cardiovascular System (Transport and Circulation);
 Integumentary System (Chemical Coordination and
 Homeostasis); Molecular Genetics (Biochemistry and
 Molecular Biophysics); Nervous System (Neural
 Coordination)

INDEX TERMS: Parts, Structures, & Systems of Organisms
 dermal diploid fibroblast: integumentary system

INDEX TERMS: Diseases
ataxia telangiectasia: genetic
 disease, immune system disease, nervous system disease,
 vascular disease, genetics

Ataxia Telangiectasia (MeSH)

INDEX TERMS: Chemicals & Biochemicals
 DNA; RNA; **ataxia telangiectasia**
 mutated [ATM]: expression; cDNA [complementary DNA];
 cyclin E-associated kinase; serine/threonine kinase;
 t-butyl-hydroperoxide

INDEX TERMS: Miscellaneous Descriptors
 DNA damage; UV radiation [ultraviolet radiation];
 oxidative stress; Meeting Abstract

REGISTRY NUMBER: 9026-43-1 (SERINE/THREONINE KINASE)

GENE NAME: human ATM gene [human **ataxia**
telangiectasia mutated gene] (Hominidae):
 expression

L214 ANSWER 21 OF 22 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
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ACCESSION NUMBER: 1999:177572 BIOSIS

DOCUMENT NUMBER: PREV199900177572

TITLE: The **ataxia telangiectasia** gene product
 is required for oxidative stress-induced G1 and G2
 checkpoint functions in human fibroblasts.

AUTHOR(S): **Shackelford, Rodney E.**; Innes, Cynthia L.;
 Sieber, Stella O.; Paules, Richard S.

CORPORATE SOURCE: Natl. Inst. Environ. Health Sci., P.O. Box 12233, Research
 Triangle Park, NC 27709, USA

SOURCE: Proceedings of the American Association for Cancer Research
 Annual Meeting, (March, 1999) Vol. 40, pp. 742. print.
 Meeting Info.: 90th Annual Meeting of the American
 Association for Cancer Research. Philadelphia,
 Pennsylvania, USA. April 10-14, 1999. American Association
 for Cancer Research.

ISSN: 0197-016X.

DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 5 May 1999
 Last Updated on STN: 5 May 1999

CONCEPT CODE: Genetics - Human 03508
 General biology - Symposia, transactions and proceedings
 00520

INDEX TERMS: Major Concepts
 Genetics

INDEX TERMS: Parts, Structures, & Systems of Organisms
 fibroblasts

INDEX TERMS: Diseases
ataxia telangiectasia: genetic
 disease, immune system disease, nervous system disease,
 vascular disease
Ataxia Telangiectasia (MeSH)

INDEX TERMS: Chemicals & Biochemicals
 reactive oxygen species; human **ataxia**
telangiectasia gene

INDEX TERMS: Miscellaneous Descriptors
 oxidative stress; G-1 checkpoint function; G-2
 checkpoint function; Meeting Abstract

REGISTRY NUMBER: 7782-44-7 (OXYGEN)

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ACCESSION NUMBER: 1997:231253 BIOSIS

DOCUMENT NUMBER: PREV199799530456
TITLE: Characterization of ATM expression in cell cycle checkpoints and cellular senescence.
AUTHOR(S): Afshari, C. A.; Innes, C. L.; Cable, P. L.; **Shackelford, R.**; Xu, G.; Hill, D.; Paules, R. S.
CORPORATE SOURCE: National Inst. Environmental Health Sciences, Research Triangle Park, NC 27709, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 157. Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research. San Diego, California, USA. April 12-16, 1997.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Jun 1997
Last Updated on STN: 9 Jul 1997
CONCEPT CODE: Cytology - Human 02508
Genetics - Human 03508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
Cardiovascular system - Blood vessel pathology 14508
Nervous system - Pathology 20506
Immunology - Immunopathology, tissue immunology 34508
INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Genetics; Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Nervous System (Neural Coordination)
INDEX TERMS: Chemicals & Biochemicals
KINASE
INDEX TERMS: Miscellaneous Descriptors
ANTIBODIES; **ATAXIA TELANGIECTASIA**; **ATAXIA TELANGIECTASIA GENE**; **ATAXIA TELANGIECTASIA MUTANT PROTEIN**; CARDIOVASCULAR SYSTEM; CELL BIOLOGY; CELL CYCLE CHECKPOINTS; CELLULAR SENESCENCE; CHROMOSOME ABNORMALITIES; EXPRESSION; FIBROBLASTS; GENETIC DISEASE; GENETICS; IMMUNE SYSTEM; IMMUNE SYSTEM DISEASE; MUTATION; NERVOUS SYSTEM DISEASE; NUCLEAR PROTEIN; PI-3 KINASE FAMILY; VASCULAR DISEASE
REGISTRY NUMBER: 9031-44-1 (KINASE)

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L20	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
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L31	24 SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L42	1665 SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/OBI
L43	2356 SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BI
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L45	8 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/BI
L46	8 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS-BAR/BI
L47	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
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L49	2363 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(ATAXIA (2A) TELANGIECT?)/BI
L50	15362 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CHELATIN AGENTS+OLD, NT/CT
L51	40989 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L31
L52	2369 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49)
L53	52491 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L50 OR L51)
L54	7 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L52 AND L53

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L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATIN AGENT)"/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATE/CN
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L23	23 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
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L31	24 SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L37	22 SEA FILE=REGISTRY ABB=ON	PLU=ON	(117-39-5/BI OR 146426-40-6/B I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR 520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR 522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
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L43	2356 SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BI

1179

1061 006

Handy 10/617943

11/06/2006

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L46	8 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS-BAR/BI
L47	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
L48	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOT?/BI
L49	2363 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(ATAXIA (2A) TELANGIECT?) /BI
L50	15362 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CHELATIN AGENTS+OLD, NT/CT
L51	40989 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L31
L52	2369 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49)
L53	52491 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L50 OR L51)
L54	7 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L52 AND L53
L55	34358 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L39
L56	4 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L54 AND L55

=> d que L71

L42	1665 SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/OBI
L43	2356 SEA FILE=HCAPLUS ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/BI
L44	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS BAR/OBI
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L46	8 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LOUIS-BAR/BI
L47	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOCUTANEOUS TELANGIECT?/BI
L48	0 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CEREBELLO OCULOT?/BI
L49	2363 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(ATAXIA (2A) TELANGIECT?) /BI
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L68	15322 SEA FILE=HCAPLUS ABB=ON	PLU=ON	HYDROXAMIC ACIDS+NT/CT
L69	29 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L68 AND L52
L70	132130 SEA FILE=HCAPLUS ABB=ON	PLU=ON	CHELAT?/BI
L71	3 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L69 AND L70

=> s (L54 or L56 or L71) not L208

L215 3 (L54 OR L56 OR L71) NOT L208

=> file medline

FILE 'MEDLINE' ENTERED AT 15:30:19 ON 17 JUL 2006

FILE LAST UPDATED: 15 JUL 2006 (20060715/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details
on the 2006 reload, enter HELP RLOAD at an arrow prompt (>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the

MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que L96

L1	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATED/CN
L12	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATED"/CN OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4 SEA FILE=REGISTRY ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI C?/CN
L23	23 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	70-51-9
L31	24 SEA FILE=REGISTRY ABB=ON	PLU=ON	L23 OR L29
L83	2457 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/CT
L84	3932 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L85	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA (2A) TELANGIECTASIA
L86	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	(L83 OR L84 OR L85)
L88	13204 SEA FILE=MEDLINE ABB=ON	PLU=ON	CHELATING AGENTS/CT
L89	92986 SEA FILE=MEDLINE ABB=ON	PLU=ON	CHELATING AGENTS+NT/CT
L90	3231 SEA FILE=MEDLINE ABB=ON	PLU=ON	IRON CHELATING AGENTS/CT
L91	19528 SEA FILE=MEDLINE ABB=ON	PLU=ON	IRON CHELATING AGENTS+NT/CT
L92	1267 SEA FILE=MEDLINE ABB=ON	PLU=ON	SIDEROPHORES/CT
L93	6055 SEA FILE=MEDLINE ABB=ON	PLU=ON	SIDEROPHORES+NT/CT
L94	SEL PLU=ON L31 1 - CHEM :		255 TERMS
L95	68933 SEA FILE=MEDLINE ABB=ON	PLU=ON	L94
L96	7 SEA FILE=MEDLINE ABB=ON	PLU=ON	L86 AND ((L88 OR L89 OR L90 OR L91 OR L92 OR L93) OR L95)

=> d que L98

L83	2457 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA/CT
L84	3932 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA TELANGIECTASIA
L85	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	ATAXIA (2A) TELANGIECTASIA
L86	3935 SEA FILE=MEDLINE ABB=ON	PLU=ON	(L83 OR L84 OR L85)
L97	37091 SEA FILE=MEDLINE ABB=ON	PLU=ON	CHELAT?

L98 5 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND L97

=> d que L105

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
 L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
 L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
 L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
 L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
 L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
 L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6)
 L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
 L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
 L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
 L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
 L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L83 2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
 L84 3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L85 3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
 L86 3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
 L88 13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT
 L89 92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT
 L90 3231 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS/CT
 L91 19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
 L92 1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT
 L93 6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT
 L94 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L95 68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
 L96 7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
 OR L91 OR L92 OR L93) OR L95)
 L100 61053 SEA FILE=MEDLINE ABB=ON PLU=ON ANTIOXID?
 L101 18939 SEA FILE=MEDLINE ABB=ON PLU=ON FLAV!NOID?/BI
 L102 32882 SEA FILE=MEDLINE ABB=ON PLU=ON FLAVONOIDS+NT/CT
 L103 QUE ABB=ON PLU=ON TRANSITION ELEMENTS+NT/CT
 L105 3 SEA FILE=MEDLINE ABB=ON PLU=ON L96 AND ((L100 OR L101 OR
 L102 OR L103))

=> d que L108

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN

L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
 L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
 L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
 L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
 L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
 L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6)
 L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
 L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
 L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
 L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
 L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/B1 OR 146426-40-6/B
 I OR 153-18-4/B1 OR 17912-87-7/B1 OR 446-72-0/B1 OR 480-16-0/B1
 OR 480-18-2/B1 OR 480-40-0/B1 OR 480-41-1/B1 OR 482-39-3/B1
 OR 490-46-0/B1 OR 491-70-3/B1 OR 491-80-5/B1 OR 50-78-2/B1 OR
 520-26-3/B1 OR 520-27-4/B1 OR 520-33-2/B1 OR 520-36-5/B1 OR
 522-12-3/B1 OR 525-82-6/B1 OR 577-85-5/B1 OR 989-51-5/B1)
 L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
 L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
 L83 2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
 L84 3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L85 3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
 L86 3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
 L88 13204 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS/CT
 L89 92986 SEA FILE=MEDLINE ABB=ON PLU=ON CHELATING AGENTS+NT/CT
 L90 3231 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS/CT
 L91 19528 SEA FILE=MEDLINE ABB=ON PLU=ON IRON CHELATING AGENTS+NT/CT
 L92 1267 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES/CT
 L93 6055 SEA FILE=MEDLINE ABB=ON PLU=ON SIDEROPHORES+NT/CT
 L94 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L95 68933 SEA FILE=MEDLINE ABB=ON PLU=ON L94
 L96 7 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND ((L88 OR L89 OR L90
 OR L91 OR L92 OR L93) OR L95)
 L106 SEL PLU=ON L39 1- CHEM : 344 TERMS
 L107 19788 SEA FILE=MEDLINE ABB=ON PLU=ON L106
 L108 2 SEA FILE=MEDLINE ABB=ON PLU=ON L107 AND L96

=> d que L119

L83 2457 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA/CT
 L84 3932 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA TELANGIECTASIA

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L85 3935 SEA FILE=MEDLINE ABB=ON PLU=ON ATAXIA (2A) TELANGIECTASIA
L86 3935 SEA FILE=MEDLINE ABB=ON PLU=ON (L83 OR L84 OR L85)
L109 QUE ABB=ON PLU=ON FERRIOXAMIN? OR DEFEROXAMIN? OR DESF
ERROXAMIN? OR DEFERRIOXAMIN?
L110 QUE ABB=ON PLU=ON EDETIC ACID/CT
L111 QUE ABB=ON PLU=ON CP94
L112 QUE ABB=ON PLU=ON HYDROXAMIC ACIDS/CT
L113 QUE ABB=ON PLU=ON APOFERRITIN/CT
L114 QUE ABB=ON PLU=ON CDTA
L115 QUE ABB=ON PLU=ON DTPA OR PENTATIC ACID
L116 QUE ABB=ON PLU=ON PENICILLAMINE
L117 QUE ABB=ON PLU=ON BATHOCUPROINE
L118 QUE ABB=ON PLU=ON BATHOCUPROIN
L119 6 SEA FILE=MEDLINE ABB=ON PLU=ON L86 AND (L109 OR L110 OR L111
OR L112 OR L113 OR L114 OR L115 OR L116 OR L117 OR L118)

=> s (L96 or L98 or L105 or L108 or L119) not L209

L216 7 (L96 OR L98 OR L105 OR L108 OR L119) NOT L209

=> file embase

FILE 'EMBASE' ENTERED AT 15:30:24 ON 17 JUL 2006
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FILE COVERS 1974 TO 17 Jul 2006 (20060717/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default)
and biweekly.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> d que L139

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6)
L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATED/CN
L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATED"/CN
OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN

L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L128 2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
 L129 3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L130 62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
 L131 2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
 L132 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
 TANEA
 L133 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
 OCULOCUTANEA
 L134 3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
 OR L132 OR L133)
 L136 98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
 L137 SEL PLU=ON L31 1 - CHEM : 255 TERMS
 L138 61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
 L139 14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)

=> d que L142

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
 L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
 L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
 L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
 L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
 L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
 L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6)
 L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
 L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
 L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
 L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
 L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L128 2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
 L129 3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L130 62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
 L131 2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA

L132 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
TANEA
L133 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
OCULOCUTANEA
L134 3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
OR L132 OR L133)
L136 98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATING AGENT+NT/CT
SEL PLU=ON L31 1- CHEM : 255 TERMS
L138 61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
L139 14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
L140 25033 SEA FILE=EMBASE ABB=ON PLU=ON FLAVONOIDS+NT/CT
L141 35447 SEA FILE=EMBASE ABB=ON PLU=ON ANTIOXIDANT+NT/CT
L142 4 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND (L140 OR L141)

=> d que L145

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6)
L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
C?/CN
L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/BI OR 146426-40-6/B
I OR 153-18-4/BI OR 17912-87-7/BI OR 446-72-0/BI OR 480-16-0/BI
OR 480-18-2/BI OR 480-40-0/BI OR 480-41-1/BI OR 482-39-3/BI
OR 490-46-0/BI OR 491-70-3/BI OR 491-80-5/BI OR 50-78-2/BI OR
520-26-3/BI OR 520-27-4/BI OR 520-33-2/BI OR 520-36-5/BI OR
522-12-3/BI OR 525-82-6/BI OR 577-85-5/BI OR 989-51-5/BI)
L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L128 2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L129 3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L130 62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L131 2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L132 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU

TANEA
L133 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
OCULOCUTANEA
L134 3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
OR L132 OR L133)
L136 98621 SEA FILE=EMBASE ABB=ON PLU=ON CHELATIN AGENT+NT/CT
SEL PLU=ON L31 1 - CHEM : 255 TERMS
L137 61283 SEA FILE=EMBASE ABB=ON PLU=ON L137
14 SEA FILE=EMBASE ABB=ON PLU=ON L134 AND (L136 OR L138)
SEL PLU=ON L39 1 - CHEM : 344 TERMS
L143 24101 SEA FILE=EMBASE ABB=ON PLU=ON L143
2 SEA FILE=EMBASE ABB=ON PLU=ON L139 AND L144

=> d que L150

L128 2332 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA+UF/CT
L129 3044 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L130 62 SEA FILE=EMBASE ABB=ON PLU=ON LOUIS BAR
L131 2 SEA FILE=EMBASE ABB=ON PLU=ON ATAXIA TELANGIECTATICA
L132 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLOOCULOCU
TANEA
L133 0 SEA FILE=EMBASE ABB=ON PLU=ON TELANGIECTASIA CEREBELLO
OCULOCUTANEA
L134 3053 SEA FILE=EMBASE ABB=ON PLU=ON (L128 OR L129 OR L130 OR L131
OR L132 OR L133)
L149 31221 SEA FILE=EMBASE ABB=ON PLU=ON CHELAT?
L150 4 SEA FILE=EMBASE ABB=ON PLU=ON L149 AND L134

=> s (L139 or L142 or L145 or L150) not L210

L217 10 (L139 OR L142 OR L145 OR L150) NOT L210

=> file biosis

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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 12 July 2006 (20060712/ED)

=> d que L168

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6)
L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATIN AGENT) "/CN
L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATED/CN

L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLADE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
 L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
 L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
 L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
 L155 3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
 L157 38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
 SEL PLU=ON L31 1 - CHEM : 255 TERMS
 L159 59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
 L160 3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
 L163 3396 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROPHOR?
 L164 42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
 L165 72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
 L166 3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
 L167 QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
 L168 7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167

=> d que L172

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
 L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
 L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
 L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
 L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
 L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
 L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6)
 L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
 L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
 L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
 L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLADE/CN
 L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
 L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLADE"/CN
 OR "DEFEROXAMINE METHANESULFONATE"/CN)
 L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
 L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
 L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
 L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
 L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
 L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTTPA/CN
 L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
 L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
 L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
 C?/CN
 L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
 OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR

L15 OR I:16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/B1 OR 146426-40-6/B
I OR 153-18-4/B1 OR 17912-87-7/B1 OR 446-72-0/B1 OR 480-16-0/B1
OR 480-18-2/B1 OR 480-40-0/B1 OR 480-41-1/B1 OR 482-39-3/B1
OR 490-46-0/B1 OR 491-70-3/B1 OR 491-80-5/B1 OR 50-78-2/B1 OR
520-26-3/B1 OR 520-27-4/B1 OR 520-33-2/B1 OR 520-36-5/B1 OR
522-12-3/B1 OR 525-82-6/B1 OR 577-85-5/B1 OR 989-51-5/B1)
L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2
L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38
L155 3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA
L157 38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
L158 SEL PLU=ON L31 1 - CHEM : 255 TERMS
L159 59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
L160 3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
L163 3396 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROPHOR?
L164 42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
L165 72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
L166 3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
L167 QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
L168 7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167
L170 SEL PLU=ON L39 1 - CHEM : 344 TERMS
L171 31206 SEA FILE=BIOSIS ABB=ON PLU=ON L170
L172 3 SEA FILE=BIOSIS ABB=ON PLU=ON L171 AND L168

=> d que L173

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN
L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN
L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN
L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN
L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN
L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN
L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6)
L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN
L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN
L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT) "/CN
L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATE/CN
L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7
L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATE"/CN
OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7
L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN
L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN
L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN
L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN
L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN
L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN
L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN
L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
C?/CN
L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)
L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9
L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29
L155 3180 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA

L157 38028 SEA FILE=BIOSIS ABB=ON PLU=ON CHELAT?
 L158 SEL PLU=ON L31 1- CHEM : 255 TERMS
 L159 59051 SEA FILE=BIOSIS ABB=ON PLU=ON L158
 L160 3182 SEA FILE=BIOSIS ABB=ON PLU=ON ATAXIA TELANGIECTASIA?
 L161 4049 SEA FILE=BIOSIS ABB=ON PLU=ON TRANSITION METAL?
 L162 160 SEA FILE=BIOSIS ABB=ON PLU=ON TRANSITION ELEM?
 L163 3396 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROPHOR?
 L164 42 SEA FILE=BIOSIS ABB=ON PLU=ON SIDEROCHROM?
 L165 72 SEA FILE=BIOSIS ABB=ON PLU=ON LOUIS BAR
 L166 3223 SEA FILE=BIOSIS ABB=ON PLU=ON L155 OR L160 OR L165
 L167 QUE ABB=ON PLU=ON L157 OR (L163 OR L164) OR L159
 L168 7 SEA FILE=BIOSIS ABB=ON PLU=ON L166 AND L167
 L173 0 SEA FILE=BIOSIS ABB=ON PLU=ON L168 AND (L161 OR L162)

=> s (L168 or L172 or L173) not L211

L218 3 (L168 OR L172 OR L173) NOT L211

=> file uspatfull

FILE 'USPATFULL' ENTERED AT 15:30:33 ON 17 JUL 2006
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 13 Jul 2006 (20060713/PD)
 FILE LAST UPDATED: 13 Jul 2006 (20060713/ED)
 HIGHEST GRANTED PATENT NUMBER: US7076805
 HIGHEST APPLICATION PUBLICATION NUMBER: US2006156447
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 REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2006
 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2006

=> d que L185

L1	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE/CN
L2	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B/CN
L3	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B C?/CN
L4	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B H?/CN
L5	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B M?/CN
L6	2 SEA FILE=REGISTRY ABB=ON	PLU=ON	FERRIOXAMINE B P?/CN
L7	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5 OR L6)
L8	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CP 94/CN
L9	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	EDTA/CN
L10	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	"EDTA (CHELATING AGENT) "/CN
L11	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DEFEROXAMINE B MESYLATED/CN
L12	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L11 AND L7
L13	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	("DEFEROXAMINE MESYLATED"/CN OR "DEFEROXAMINE METHANESULFONATE"/CN)
L14	0 SEA FILE=REGISTRY ABB=ON	PLU=ON	L13 AND L7
L15	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL/CN
L16	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DESFERAL M?/CN
L17	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	APOFERRITIN?/CN
L18	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	CDTA/CN
L19	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	DTPA/CN
L20	1 SEA FILE=REGISTRY ABB=ON	PLU=ON	PENICILLAMINE/CN
L21	6 SEA FILE=REGISTRY ABB=ON	PLU=ON	BATHOCUP?/CN
L22	4 SEA FILE=REGISTRY ABB=ON	PLU=ON	DIETHYLENETRIAMINE PENTAACETI

C?/CN

L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)

L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9

L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29

L183 5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31

L184 2355 SEA FILE=USPATFULL ABB=ON PLU=ON ATAXIA TELANGIECTASIA

L185 6 SEA FILE=USPATFULL ABB=ON PLU=ON L183 AND L184

=> d que L187

L1 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE/CN

L2 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B/CN

L3 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B C?/CN

L4 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B H?/CN

L5 1 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B M?/CN

L6 2 SEA FILE=REGISTRY ABB=ON PLU=ON FERRIOXAMINE B P?/CN

L7 6 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6)

L8 1 SEA FILE=REGISTRY ABB=ON PLU=ON CP 94/CN

L9 1 SEA FILE=REGISTRY ABB=ON PLU=ON EDTA/CN

L10 1 SEA FILE=REGISTRY ABB=ON PLU=ON "EDTA (CHELATING AGENT)"/CN

L11 1 SEA FILE=REGISTRY ABB=ON PLU=ON DEFEROXAMINE B MESYLATED/CN

L12 0 SEA FILE=REGISTRY ABB=ON PLU=ON L11 AND L7

L13 1 SEA FILE=REGISTRY ABB=ON PLU=ON ("DEFEROXAMINE MESYLATED"/CN
OR "DEFEROXAMINE METHANESULFONATE"/CN)

L14 0 SEA FILE=REGISTRY ABB=ON PLU=ON L13 AND L7

L15 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL/CN

L16 1 SEA FILE=REGISTRY ABB=ON PLU=ON DESFERAL M?/CN

L17 1 SEA FILE=REGISTRY ABB=ON PLU=ON APOFERRITIN?/CN

L18 1 SEA FILE=REGISTRY ABB=ON PLU=ON CDTA/CN

L19 1 SEA FILE=REGISTRY ABB=ON PLU=ON DTPA/CN

L20 1 SEA FILE=REGISTRY ABB=ON PLU=ON PENICILLAMINE/CN

L21 6 SEA FILE=REGISTRY ABB=ON PLU=ON BATHOCUP?/CN

L22 4 SEA FILE=REGISTRY ABB=ON PLU=ON DIETHYLENETRIAMINE PENTAACETI
C?/CN

L23 23 SEA FILE=REGISTRY ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5
OR L6 OR L7 OR L8 OR L9 OR L10 OR L11 OR L12 OR L13 OR L14 OR
L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22)

L29 1 SEA FILE=REGISTRY ABB=ON PLU=ON 70-51-9

L31 24 SEA FILE=REGISTRY ABB=ON PLU=ON L23 OR L29

L37 22 SEA FILE=REGISTRY ABB=ON PLU=ON (117-39-5/B1 OR 146426-40-6/B
I OR 153-18-4/B1 OR 17912-87-7/B1 OR 446-72-0/B1 OR 480-16-0/B1
OR 480-18-2/B1 OR 480-40-0/B1 OR 480-41-1/B1 OR 482-39-3/B1
OR 490-46-0/B1 OR 491-70-3/B1 OR 491-80-5/B1 OR 50-78-2/B1 OR
520-26-3/B1 OR 520-27-4/B1 OR 520-33-2/B1 OR 520-36-5/B1 OR
522-12-3/B1 OR 525-82-6/B1 OR 577-85-5/B1 OR 989-51-5/B1)

L38 1 SEA FILE=REGISTRY ABB=ON PLU=ON 50-78-2

L39 21 SEA FILE=REGISTRY ABB=ON PLU=ON L37 NOT L38

L183 5870 SEA FILE=USPATFULL ABB=ON PLU=ON L31

L184 2355 SEA FILE=USPATFULL ABB=ON PLU=ON ATAXIA TELANGIECTASIA

L185 6 SEA FILE=USPATFULL ABB=ON PLU=ON L183 AND L184

L186 1795 SEA FILE=USPATFULL ABB=ON PLU=ON L39

L187 1 SEA FILE=USPATFULL ABB=ON PLU=ON L185 AND L186

=> s (L185 or L187) not l212

L219 5 (L185 OR L187) NOT L212

=> file wpix

FILE 'WPIX' ENTERED AT 15:30:37 ON 17 JUL 2006
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FILE LAST UPDATED: 14 JUL 2006 <20060714/UP>
MOST RECENT DERWENT UPDATE: 200645 <200645/DW>
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INDEX ENHANCEMENTS PLEASE VISIT:
[<http://www.stn-international.de/stndatabases/details/dwpi_r.html><<<](http://www.stn-international.de/stndatabases/details/dwpi_r.html)
'BIX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE

=> d que L197

L188 247 SEA FILE=WPIX ABB=ON PLU=ON ATAXIA TELANGIECTASIA/BIX
L195 5533 SEA FILE=WPIX ABB=ON PLU=ON (RAAMBT/DCN OR RAGNQ8/DCN OR
RA0DFA/DCN OR RAOEMC/DCN OR RA0JBK/DCN OR RA00TF/DCN OR
RA0055/DCN OR RA021P/DCN OR RA0529/DCN OR RA1HHQ/DCN OR
RA1XA5/DCN OR RA37W9/DCN OR R00064/DCN OR R00195/DCN OR
R00268/DCN OR R00971/DCN OR R01179/DCN OR R01318/DCN OR
R01319/DCN OR R03811/DCN OR R03812/DCN OR R03949/DCN OR
R04870/DCN OR R06069/DCN OR R06174/DCN OR R06413/DCN OR
R06747/DCN OR R07001/DCN OR R07027/DCN OR R08105/DCN OR
R08504/DCN OR R09163/DCN OR R09222/DCN OR R09884/DCN OR
R11605/DCN OR R19085/DCN OR R19452/DCN OR R20811/DCN OR
R22037/DCN)
L197 2 SEA FILE=WPIX ABB=ON PLU=ON L188 AND L195

=> s L197 not L213

L220 2 L197 NOT L213

=> => dup rem L215 L216 L217 L218 L219 L220
FILE 'HCAPLUS' ENTERED AT 15:31:21 ON 17 JUL 2006
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FILE 'WPIX' ENTERED AT 15:31:21 ON 17 JUL 2006
 COPYRIGHT (C) 2006 THE THOMSON CORPORATION
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 PROCESSING COMPLETED FOR L217
 PROCESSING COMPLETED FOR L218
 PROCESSING COMPLETED FOR L219
 PROCESSING COMPLETED FOR L220
 L221 24 DUP REM L215 L216 L217 L218 L219 L220 (6 DUPLICATES REMOVED)
 ANSWERS '1-3' FROM FILE HCAPLUS
 ANSWERS '4-9' FROM FILE MEDLINE
 ANSWERS '10-16' FROM FILE EMBASE
 ANSWER '17' FROM FILE BIOSIS
 ANSWERS '18-22' FROM FILE USPATFULL
 ANSWERS '23-24' FROM FILE WPIX

=> d ibib abs hitind hitstr L221 1-3; d iall L221 4-17; d ibib abs kwic hitstr L221
 18-22; d iall ind L221 23-24

L221 ANSWER 1 OF 24 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
 ACCESSION NUMBER: 2005:1214439 HCAPLUS
 DOCUMENT NUMBER: 144:18854
 TITLE: Tachpyridine, a metal chelator, induces G2 cell-cycle
 arrest, activates checkpoint kinases, and sensitizes
 cells to ionizing radiation
 AUTHOR(S): Turner, JoLyn; Koumenis, Constantinos; Kute, Timothy
 E.; Planalp, Roy P.; Brechbiel, Martin W.; Beardsley,
 Dillon, Cody, Brooke; Brown, Kevin D.; Torti, Frank
 M.; Torti, Suzy V.
 CORPORATE SOURCE: Department of Biochemistry, Wake Forest University
 Health Sciences, Durham, NH, USA
 SOURCE: Blood (2005), 106(9), 3191-3199
 CODEN: BLOOAW; ISSN: 0006-4971
 PUBLISHER: American Society of Hematology
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Iron is critical for cell growth and proliferation. Iron chelators are being
 explored for a number of clin. applications, including the treatment of
 neurodegenerative disorders, heart disease, and cancer. To uncover
 mechanisms of action of tachpyridine, a chelator currently undergoing
 preclin. evaluation as an anticancer agent, cell-cycle anal. was
 performed. Tachpyridine arrested cells at G2, a radiosensitive phase of
 the cell cycle, and enhanced the sensitivity of cancer cells but not
 nontransformed cells to ionizing radiation. G2 arrest was p53 independent
 and was accompanied by activation of the checkpoint kinases CHK1 and CHK2.
 G2 arrest was blocked by UCN-01, a CHK1 inhibitor, but proceeded in CHK2
 knock-out cells, indicating a critical role for CHK1 in G2 arrest.
 Tachpyridine-induced cell-cycle arrest was abrogated in cells treated with
 caffeine, an inhibitor of the *ataxia-telangiectasia*
 mutated/*ataxia-telangiectasia*-mutated and Rad3-related
 (ATM/ATR) kinases. Further, G2 arrest proceeded in ATM-deficient cells
 but was blocked in ATR-deficient cells, implicating ATR as the proximal
 kinase in tachpyridine-mediated G2 arrest. Collectively, our results
 suggest that iron chelators may function as antitumor and radioenhancing
 agents and uncover a previously unexplored activity of iron chelators in

activation of ATR and checkpoint kinases.

CC 8-9 (Radiation Biochemistry)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ATM (*ataxia telangiectasia* mutated); metal chelator tachpyridine as antitumor radiosensitizer and effect on cell cycle and checkpoint kinases)

IT Antitumor agents

Chelating agents

Human

Radiosensitizers, biological

Radiotherapy

(metal chelator tachpyridine as antitumor radiosensitizer and effect on cell cycle and checkpoint kinases)

REFERENCE COUNT: 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L221 ANSWER 2 OF 24 HCPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:266901 HCPLUS

DOCUMENT NUMBER: 140:302341

TITLE: Protein complexes of the tumor necrosis factor- α signalling pathway for diagnosis, therapy and drug screening

INVENTOR(S): Bouwmeester, Tewis; Huhse, Bettina; Bauch, Angela; Ruffner, Heinz; Bauer, Andreas; Kruse, Ulrich; Kuester, Bernhard; Superti-Furga, Giulio

PATENT ASSIGNEE(S): Cellzome Ag, Germany

SOURCE: Eur. Pat. Appl., 549 pp.

CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

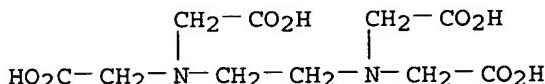
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1403282	A1	20040331	EP 2002-21809	20020926
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
WO 2004035783	A2	20040429	WO 2003-EP50655	20030924
WO 2004035783	C2	20040930		
WO 2004035783	A3	20041111		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003298261	A1	20040504	AU 2003-298261	20030924
PRIORITY APPLN. INFO.:			EP 2002-21809	A 20020926
			EP 2003-100274	A 20030210
			WO 2003-EP50655	W 20030924

AB The present invention relates to protein complexes of the Tumor necrosis factor- α -signaling pathway, component proteins of said complexes, fragments and derivs. of the component proteins and antibodies specific to the complexes. The present invention also relates to methods for use of

the complexes of the TNF- α -signaling pathway and their interaction in screening, diagnosis and therapy as well to methods of preparing the complexes. Pharmaceutical compns. comprising the protein complexes and antibodies specific to the complexes are especially useful for diagnosis and treatment of inflammation, infection, neurodegenerative disease and cancer.

IC ICM C07K014-705
 ICS C07K014-715; C07K016-28; C07K017-00; A61K038-17; G01N033-50
 CC 15-5 (Immunochemistry)
 Section cross-reference(s): 1, 3, 9, 63
 IT Proteins
 RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
 (ataxia-telangiectasia group D-associated; protein complexes of the tumor necrosis factor- α signalling pathway and antibodies for drug screening and for diagnosis and therapy)
 IT 60-00-4, EDTA, biological studies 75-12-7, Formamide, biological studies 1185-53-1, Tris hydrochloride 9003-39-8, PVP 9042-14-2, Dextran sulfate
 RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
 (protein complexes of the tumor necrosis factor- α signalling pathway and antibodies for drug screening and for diagnosis and therapy of inflammation, infection, neurodegenerative disease and cancer)
 IT 60-00-4, EDTA, biological studies
 RL: ARU (Analytical role, unclassified); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
 (protein complexes of the tumor necrosis factor- α signalling pathway and antibodies for drug screening and for diagnosis and therapy of inflammation, infection, neurodegenerative disease and cancer)
 RN 60-00-4 HCAPLUS
 CN Glycine, N,N'-1,2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L221 ANSWER 3 OF 24 HCAPLUS COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 2002:397930 HCAPLUS
 DOCUMENT NUMBER: 136:374807
 TITLE: Cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid
 INVENTOR(S): Gianfranco de Paoli, Ambrosi
 PATENT ASSIGNEE(S): General Topics S.R.L., Italy
 SOURCE: Ital., 20 pp.
 CODEN: ITXXBY
 DOCUMENT TYPE: Patent
 LANGUAGE: Italian
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
IT 1299623	B1	20000324	IT 1998-BS10	19980223

PRIORITY APPLN. INFO.:

IT 1998-BS10

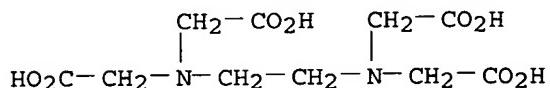
19980223

- AB The invention concerns a composition for cosmetic or pharmaceutical use which contains as active ingredients at least lipoic acid (both reduced form and dehydrolipoic acid) and pyruvic acid, their salts, esters, and amides and stereoisomers. Each may be present in amts. from 0.0001 to 90% weight/weight
- IC ICM A61K
- CC 63-6 (Pharmaceuticals)
- Section cross-reference(s): 62
- IT Nervous system, disease
 (ataxia telangiectasia; cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)
- IT 50-21-5, 2-Hydroxypropanoic acid, biological studies 50-81-7, Ascorbic acid, biological studies 52-90-4, Cysteine, biological studies 56-40-6, Glycine, biological studies 56-41-7, Alanine, biological studies 56-45-1, Serine, biological studies 56-84-8, Aspartic acid, biological studies 56-85-9, Glutamine, biological studies 56-86-0, Glutamic acid, biological studies 56-87-1, Lysine, biological studies 57-10-3, Palmitic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 58-85-5, Biotin 59-30-3, Folic acid, biological studies 59-43-8, Thiamine, biological studies 59-67-6, Niacin, biological studies 60-00-4, Ethylenediaminetetraacetic acid, biological studies 60-18-4, Tyrosine, biological studies 60-33-3, Linoleic acid, biological studies 61-90-5, Leucine, biological studies 63-68-3, Methionine, biological studies 63-91-2, Phenylalanine, biological studies 65-23-6, Pyridoxine 66-72-8, Pyridoxal 68-26-8, Retinol 69-72-7, 2-Hydroxybenzoic acid, biological studies 70-47-3, Asparagine, biological studies 71-00-1, Histidine, biological studies 72-18-4, Valine, biological studies 72-19-5, Threonine, biological studies 73-22-3, Tryptophan, biological studies 73-32-5, Isoleucine, biological studies 74-79-3, Arginine, biological studies 79-14-1, Hydroxyethanoic acid, biological studies 79-83-4, Pantothenic acid 81-13-0, Panthenol 83-88-5, Riboflavin, biological studies 87-69-4, 2,3-Dihydroxybutanedioic acid, biological studies 98-92-0, Nicotinamide 105-45-3 107-35-7, Taurine 112-80-1, Oleic acid, biological studies 114-07-8, Erythromycin 116-31-4, Retinaldehyde 123-31-9, Hydroquinone, biological studies 127-17-3, Pyruvic acid, biological studies 127-17-3D, Pyruvic acid, derivs. 141-97-9 143-07-7, Lauric acid, biological studies 147-85-3, Proline, biological studies 150-13-0 153-18-4, Rutin 302-79-4, Retinoic acid 373-49-9, Palmitoleic acid 443-48-1, Metronidazol 463-40-1, Linolenic acid 464-92-6, Asiatic acid 473-81-4, 2,3-Dihydroxypropanoic acid 506-32-1, Arachidonic acid 526-95-4, Gluconic acid 541-50-4, biological studies 544-63-8, Myristic acid, biological studies 544-64-9, Myristoleic acid 557-59-5, Lignoceric acid 600-15-7, 2-Hydroxybutanoic acid 600-22-6 617-35-6 693-72-1, Vaccenic acid 1200-22-2, Lipoic acid 1200-22-2D, Lipoic acid, derivs. 3380-34-5, Tricosan 3416-24-8, Glucosamine 6556-12-3, Glucuronic acid 7235-40-7, β Carotene 7512-17-6, Acetylglucosamine 9004-61-9, Hyaluronic acid 10118-90-8, Minocycline 16830-15-2, Asiaticoside 18323-44-9, Clindamycin 18449-41-7, Madecassic acid 29204-02-2, Gadoleic acid 34540-22-2, Madecassoside 38882-78-9 55306-03-1, Sericic acid 55306-04-2, Sericoside RL: COS (Cosmetic use); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
 (cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)
- IT 60-00-4, Ethylenediaminetetraacetic acid, biological studies 153-18-4, Rutin
 RL: COS (Cosmetic use); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study);

PROC (Process); USES (Uses)
 (cosmetic or pharmaceutical composition based on lipoic acid and pyruvic acid)

RN 60-00-4 HCPLUS

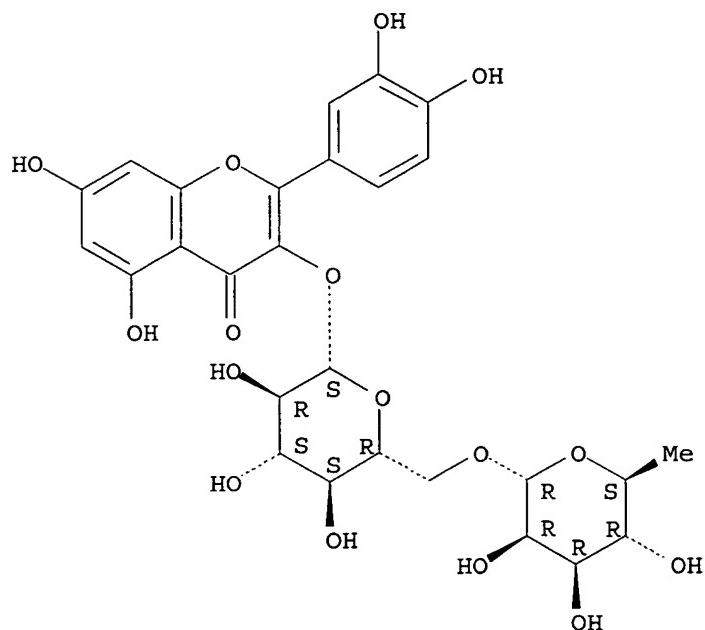
CN Glycine, N,N'-1,2-ethanediylbis[N-(carboxymethyl)- (9CI) (CA INDEX NAME)



RN 153-18-4 HCPLUS

CN 4H-1-Benzopyran-4-one, 3-[[6-O-(6-deoxy- α -L-mannopyranosyl)- β -D-glucopyranosyl]oxy]-2-(3,4-dihydroxyphenyl)-5,7-dihydroxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L221 ANSWER 4 OF 24

MEDLINE on STN

DUPLICATE 1

ACCESSION NUMBER: 2006242510 IN-PROCESS

DOCUMENT NUMBER: PubMed ID: 16651613

TITLE: SUMOylation attenuates sensitivity toward hypoxia- or desferroxamine-induced injury by modulating adaptive responses in salivary epithelial cells.

AUTHOR: Nguyen Ha-Van; Chen Jo-Lin; Zhong Jenny; Kim Kwang-Jin; Crandall Edward D; Borok Zea; Chen Yuan; Ann David K

CORPORATE SOURCE: Department of Molecular Pharmacology and Toxicology, University of Southern California, Los Angeles 90033-1049, USA.

CONTRACT NUMBER: CA-94595 (NCI)
 HL-38578 (NHLBI)

HL-38621 (NHLBI)
 HL-38658 (NHLBI)
 HL-62569 (NHLBI)
 HL-64365 (NHLBI)
 R01-DE-10742 (NIDCR)
 R01-DE-14183 (NIDCR)

SOURCE: The American journal of pathology, (2006 May) Vol. 168, No. 5, pp. 1452-63.
 Journal code: 0370502. ISSN: 0002-9440.

PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals
 ENTRY DATE: Entered STN: 3 May 2006
 Last Updated on STN: 23 May 2006

ABSTRACT:
 Hypoxic stress activates various signal transduction pathways including posttranslational modification with the ubiquitin-like SUMO protein (SUMOylation). However, the molecular mechanisms by which SUMOylation regulates hypoxic responses remain unclear. Here, we investigated the ability of rat salivary Pa-4 epithelial cells to resist cell injury elicited by 1% O₂- or hypoxia-mimetic desferroxamine (DFO)-stimulated SUMOylation processes. By using Pa-4 cells stably transduced with lenti-SUMO-1 and a cell-permeant peptide harboring SUMO-binding motif to interfere with SUMO-dependent protein-protein interactions, we demonstrate that SUMOylation augments cell survival against DFO treatment. This appeared to be partly mediated through attenuation of Protein Kinase C (PKC)-delta activation and caspase-3 cleavage, hallmarks of pro-apoptotic signaling. Intriguingly, DFO-induced phosphorylation of DNA damage marker **ataxia-telangiectasia**-mutated protein S1981 preceded activation of PKCdelta and caspase-3. Constitutive SUMOylation facilitated 1% O₂- or DFO-induced nuclear factor kappaB transactivation, possibly via activation of genotoxic signaling cascade. In addition, we observed transient preservation of transepithelial electrical resistance during the early stage of hypoxia (1% O₂) as well as enhanced transepithelial electrical resistance recovery after prolonged hypoxia in SUMO-1-expressing cell monolayers. In conclusion, our results unveil a previously unrecognized mechanism by which SUMOylation and activation of **ataxia-telangiectasia**-mutated protein, PKCdelta, caspase-3, and nuclear factor kappaB signaling pathways modulate salivary adaptive responses to stress in cells exposed to either 1% O₂ or DFO.

L221 ANSWER 5 OF 24 MEDLINE on STN DUPLICATE 3
 ACCESSION NUMBER: 97433222 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9288891
 TITLE: Use of a postlabelling assay to examine the removal of radiation-induced DNA lesions by purified enzymes and human cell extracts.
 AUTHOR: Weinfeld M; Lee J; Ruiqi G; Karimi-Busheri F; Chen D; Allalunis-Turner J
 CORPORATE SOURCE: Department of Oncology, University of Alberta, Cross Cancer Institute, Edmonton, Canada.. mweinfel@gpu.srv.ualberta.ca
 SOURCE: Mutation research, (1997 Aug 1) Vol. 378, No. 1-2, pp. 127-37.
 Journal code: 0400763. ISSN: 0027-5107.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals

ENTRY MONTH: 199709
 ENTRY DATE: Entered STN: 8 Oct 1997
 Last Updated on STN: 8 Oct 1997
 Entered Medline: 25 Sep 1997

ABSTRACT:

We have used a ³²P-postlabelling assay to examine the activity of purified *Escherichia coli* endonuclease IV, human apurinic/apyrimidinic endonuclease I and human cell-free extracts towards irradiated DNA. The assay can detect thymine glycols, 3'-phosphoglycolate groups and at least one other major lesion that has yet to be fully characterized. It was observed that endonuclease IV removed the phosphoglycolates and the uncharacterized lesion(s) suggesting that the latter are abasic sites with modified deoxyribose residues. The purified human enzyme acted only on the phosphoglycolate residues. Cell-free extract, prepared from A549 lung carcinoma cells by sonication or treatment with toluene, efficiently removed the phosphoglycolate and unknown lesions, but was less reactive towards thymine glycols. The extract was completely inactivated by heating at 60 degrees C for 10 min. Removal of the unknown product and phosphoglycolate did not require magnesium, but 1 mM **EDTA** did inhibit release of the latter. The cell-free extract exhibited substantially more activity towards native than heat-denatured DNA. A comparison of extracts prepared from 4 cell lines displaying a range of radiosensitivities, including an *ataxia telangiectasia* cell line, showed that all contained similar levels of repair activity towards the detectable lesions.

CONTROLLED TERM: Cell Extracts

Cell Survival

*DNA: ME, metabolism

DNA: RE, radiation effects

*DNA Damage

*DNA Repair

DNA, Single-Stranded: ME, metabolism

DNA- (Apurinic or Apyrimidinic Site) Lyase

*Deoxyribonuclease I: ME, metabolism

Deoxyribonuclease IV (Phage T4-Induced)

Electrophoresis, Polyacrylamide Gel

Escherichia coli: EN, enzymology

**Escherichia coli* Proteins

Gamma Rays: AE, adverse effects

Glycolates: ME, metabolism

Humans

*Lyases: ME, metabolism

Magnesium: PD, pharmacology

Nucleic Acid Denaturation

Phosphorus Radioisotopes: ME, metabolism

Research Support, Non-U.S. Gov't

Thymine: AA, analogs & derivatives

Thymine: ME, metabolism

Tumor Cells, Cultured

CAS REGISTRY NO.: 13147-57-4 (phosphoglycolate); 2943-56-8 (thymine glycol); 65-71-4 (Thymine); 7439-95-4 (Magnesium); 9007-49-2 (DNA)

CHEMICAL NAME: 0 (Cell Extracts); 0 (DNA, Single-Stranded); 0 (*Escherichia coli* Proteins); 0 (Glycolates); 0 (Phosphorus Radioisotopes); EC 3.1.21.1 (Deoxyribonuclease I); EC 3.1.21.2 (Deoxyribonuclease IV (Phage T4-Induced)); EC 3.1.21.2 (endonuclease IV, *E coli*); EC 4. (Lyases); EC 4.2.99.18 (DNA-(Apurinic or Apyrimidinic Site) Lyase)

L221 ANSWER 6 OF 24 MEDLINE on STN

ACCESSION NUMBER: 2004083906 MEDLINE

DOCUMENT NUMBER: PubMed ID: 14716295

TITLE: The EBNA-3 gene family proteins disrupt the G2/M

checkpoint.

AUTHOR: Krauer Kenia G; Burgess Andrew; Buck Marion; Flanagan James; Sculley Tom B; Gabrielli Brian

CORPORATE SOURCE: Queensland Institute of Medical Research and Joint Oncology Program, University of Queensland, Brisbane, Australia.. keniaK@qimr.edu.au

SOURCE: Oncogene, (2004 Feb 19) Vol. 23, No. 7, pp. 1342-53.
Journal code: 8711562. ISSN: 0950-9232.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200403

ENTRY DATE: Entered STN: 20 Feb 2004
Last Updated on STN: 11 Mar 2004
Entered Medline: 10 Mar 2004

ABSTRACT:

The Epstein-Barr nuclear antigens (EBNA), EBNA-3, -4 and -6, have previously been shown to act as transcriptional regulators, however, this study identifies another function for these proteins, disruption of the G2/M checkpoint. Lymphoblastoid cell lines (LCLs) treated with a G2/M initiating drug azelaic bishydroxamine (ABHA) did not show a G2/M checkpoint response, but rather they display an increase in cell death, a characteristic of sensitivity to the cytotoxic effects of the drug. Cell cycle analysis demonstrated that the individual expression of EBNA-3, -4 or -6 are capable of disrupting the G2/M checkpoint response induced by ABHA resulting in increased toxicity, whereas EBNA-2, and -5 were not. EBNA-3 gene family protein expression also disrupted the G2/M checkpoint initiated in response to the genotoxin etoposide and the S phase inhibitor hydroxyurea. The G2 arrest in response to these drugs were sensitive to caffeine, suggesting that ATM/ATR signalling in these checkpoint responses may be blocked by the EBNA-3 family proteins. The function of EBNA-3, -4 and -6 proteins appears to be more complex than anticipated and these data suggest a role for these proteins in disrupting the host cell cycle machinery.

CONTROLLED TERM: *Cell Cycle Proteins
DNA Damage: PH, physiology
DNA-Binding Proteins
Epstein-Barr Virus Nuclear Antigens: IM, immunology
*Epstein-Barr Virus Nuclear Antigens: ME, metabolism
G2 Phase: DE, drug effects
*G2 Phase: PH, physiology
Histone Deacetylases: AI, antagonists & inhibitors
Humans

Hydroxamic Acids: PD, pharmacology

Mitosis: DE, drug effects

*Mitosis: PH, physiology

Precipitin Tests

Protein-Serine-Threonine Kinases: IM, immunology

Protein-Serine-Threonine Kinases: ME, metabolism

Research Support, Non-U.S. Gov't

Signal Transduction: PH, physiology

Tumor Suppressor Proteins

CAS REGISTRY NO.: 18992-11-5 (azelaic bishydroxamic acid)

CHEMICAL NAME: 0 (Cell Cycle Proteins); 0 (DNA-Binding Proteins); 0 (Epstein-Barr Virus Nuclear Antigens); 0 (Hydroxamic Acids); 0 (Tumor Suppressor Proteins); EC 2.7.1.- (ATR protein, human); EC 2.7.1.37 (Protein-Serine-Threonine Kinases); EC 2.7.1.37 (*ataxia telangiectasia* mutated protein); EC 2.7.1.37 (checkpoint kinase 2); EC 3.5.1.- (Histone Deacetylases)

L221 ANSWER 7 OF 24 MEDLINE on STN
ACCESSION NUMBER: 2003256335 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12782595
TITLE: Histone deacetylase inhibitors activate p21(WAF1)
expression via ATM.
AUTHOR: Ju Rong; Muller Mark T
CORPORATE SOURCE: Department of Molecular Genetics, The Ohio State
University, Columbus, Ohio 43210, USA.
SOURCE: Cancer research, (2003 Jun 1) Vol. 63, No. 11, pp. 2891-7.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 4 Jun 2003
Last Updated on STN: 1 Aug 2003
Entered Medline: 31 Jul 2003

ABSTRACT:
Histone deacetylase (HDAC) inhibitors are known to induce expression of genes such as p21(WAF1), thereby, leading to cell cycle arrest. In this work, we show that p21(WAF1) induction by HDAC inhibitors (depsipeptide and trichostatin A) is defective in *Ataxia telangiectasia* (AT) cells but normal in matched wild-type (WT) cells (human diploid fibroblasts). To verify the role of ATM in this effect, we show that ectopic expression of the WT ATM gene in an AT cell line fully restores p21(WAF1) induction by the HDAC inhibitors. Furthermore, because caffeine and wortmannin attenuate p21(WAF1) induction in WT cells, it is probable that the phosphatidylinositol 3'-kinase activity is essential for this process. Besides the p21(WAF1) promoter, activation of topoisomerase IIIalpha and SV40 promoters by the HDAC inhibitors are also decreased in the AT cell lines relative to WT cells; thus, these findings pertain to other promoters. Finally, despite the obvious induction deficiency of gene expression, the overall levels of H3 and H4 histone acetylation appear to be the same between AT and normal cells in response to HDAC inhibitor treatments. Taken together, the data indicate that ATM is involved in histone acetylation-mediated gene regulation.

CONTROLLED TERM: 1-Phosphatidylinositol 3-Kinase: ME, metabolism
Acetylation
Ataxia Telangiectasia: PA, pathology
Cell Cycle Proteins
Cyclin-Dependent Kinase Inhibitor p21
*Cyclins: BI, biosynthesis
Cyclins: GE, genetics
DNA-Binding Proteins
*Depsipeptides
Enzyme Inhibitors: PD, pharmacology
Gene Expression Regulation: PH, physiology
*Histone Deacetylases: AI, antagonists & inhibitors
Histones: GE, genetics
Histones: ME, metabolism
Humans
Hydroxamic Acids: PD, pharmacology
Peptides, Cyclic: PD, pharmacology
Phosphorylation
Promoter Regions (Genetics): DE, drug effects
Protein-Serine-Threonine Kinases: BI, biosynthesis
Protein-Serine-Threonine Kinases: GE, genetics
*Protein-Serine-Threonine Kinases: PH, physiology
Transfection

Tumor Suppressor Proteins

CAS REGISTRY NO.: 128517-07-7 (FR 901228); 58880-19-6 (trichostatin A)
CHEMICAL NAME: 0 (CDKN1A protein, human); 0 (Cell Cycle Proteins); 0
(Cyclin-Dependent Kinase Inhibitor p21); 0 (Cyclins); 0
(DNA-Binding Proteins); 0 (Depsipeptides); 0 (Enzyme
Inhibitors); 0 (Histones); 0 (Hydroxamic Acids); 0
(Peptides, Cyclic); 0 (Tumor Suppressor Proteins); EC
2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.1.37
(Protein-Serine-Threonine Kinases); EC 2.7.1.37
(ataxia telangiectasia mutated protein);
EC 3.5.1.- (Histone Deacetylases)

L221 ANSWER 8 OF 24 MEDLINE on STN
ACCESSION NUMBER: 97459668 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9315628
TITLE: Regulation of p53 by metal ions and by **antioxidants**
: dithiocarbamate down-regulates p53 DNA-binding activity
by increasing the intracellular level of copper.
AUTHOR: Verhaegh G W; Richard M J; Hainaut P
CORPORATE SOURCE: Unit of Mechanisms of Carcinogenesis, International Agency
for Research on Cancer, Lyon, France.
SOURCE: Molecular and cellular biology, (1997 Oct) Vol. 17, No. 10,
pp. 5699-706.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 5 Nov 1997
Last Updated on STN: 17 Dec 2002
Entered Medline: 23 Oct 1997

ABSTRACT:
Mutations in the p53 tumor suppressor gene frequently fall within the specific DNA-binding domain and prevent the molecule from transactivating normal targets. DNA-binding activity is regulated in vitro by metal ions and by redox conditions, but whether these factors also regulate p53 in vivo is unclear. To address this question, we have analyzed the effect of pyrrolidine dithiocarbamate (PDTC) on p53 DNA-binding activity in cell lines expressing wild-type p53. PDTC is commonly regarded as an **antioxidant**, but it can also bind and transport external copper ions into cells and thus exert either pro- or **antioxidant** effects in different situations. We report that PDTC, but not N-acetyl-L-cysteine, down-regulated the specific DNA-binding activity of p53. Loss of DNA binding correlated with disruption of the immunologically "wild-type" p53 conformation. Using different ***chelators*** to interfere with copper transport by PDTC, we found that ***bathocuproinedisulfonic*** acid (BCS), a non-cell-permeable **chelator** of Cu⁺, prevented both copper import and p53 down-regulation. In contrast, 1,10-orthophenanthroline, a cell-permeable ***chelator*** of Cu²⁺, promoted the redox activity of copper and up-regulated p53 DNA-binding activity through a DNA damage-dependent pathway. We have previously reported that p53 protein binds copper in vitro in the form of Cu⁺ (P. Hainaut, N. Rolley, M. Davies, and J. Milner, Oncogene 10:27-32, 1995). The data reported here indicate that intracellular levels and redox activity of copper are critical for p53 protein conformation and DNA-binding activity and suggest that copper ions may participate in the physiological control of p53 function.

CONTROLLED TERM: Acetylcysteine: PD, pharmacology
*Antioxidants: PD, pharmacology
Cell Cycle

Cell Cycle Proteins
 Cell Line
 **Che*lating Agents: PD, pharmacology
 **Copper*: ME, metabolism
 Cyclin-Dependent Kinase Inhibitor p21
 Cyclins: BI, biosynthesis
 DNA: ME, metabolism
 DNA Damage
 DNA-Binding Proteins
 Humans
 Hydrogen Peroxide: PD, pharmacology
 Intercalating Agents: PD, pharmacology
 Ion Transport: DE, drug effects
 Lipid Peroxidation
 Oxidation-Reduction
 Oxidative Stress
 Phenanthrolines: PD, pharmacology
 Protein Binding: DE, drug effects
 Protein Conformation: DE, drug effects
 *Protein-Serine-Threonine Kinases
 Proteins: PH, physiology
 Pyrrolidines: PK, pharmacokinetics
 Research Support, Non-U.S. Gov't
 Thiocarbamates: PK, pharmacokinetics
 *Thiocarbamates: PD, pharmacology
 Tumor Cells, Cultured
 Tumor Suppressor Protein p53: BI, biosynthesis
 Tumor Suppressor Protein p53: CH, chemistry
 Tumor Suppressor Protein p53: DE, drug effects
 *Tumor Suppressor Protein p53: ME, metabolism
 Tumor Suppressor Proteins
 CAS REGISTRY NO.: 14708-99-7 (ferroin); 25769-03-3 (pyrrolidine dithiocarbamic acid); 616-91-1 (Acetylcysteine); 73348-75-1 (bathocuproine sulfonate); 7440-50-8 (Copper); 7722-84-1 (Hydrogen Peroxide); 9007-49-2 (DNA)
 CHEMICAL NAME: 0 (Antioxidants); 0 (CDKN1A protein, human); 0 (Cell Cycle Proteins); 0 (Che*lating* Agents); 0 (Cyclin-Dependent Kinase Inhibitor p21); 0 (Cyclins); 0 (DNA-Binding Proteins); 0 (Intercalating Agents); 0 (Phenanthrolines); 0 (Proteins); 0 (Pyrrolidines); 0 (Thiocarbamates); 0 (Tumor Suppressor Protein p53); 0 (Tumor Suppressor Proteins); EC 2.7.1.37 (Protein-Serine-Threonine Kinases); EC 2.7.1.37 (ataxia telangiectasia mutated protein)

L221 ANSWER 9 OF 24 MEDLINE on STN
 ACCESSION NUMBER: 84002886 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 6616960
 TITLE: Abnormalities of lymphocyte locomotion in immunodeficiency disease.
 AUTHOR: Van Epps D E; El-Naggar A; Ochs H D
 CONTRACT NUMBER: AI-07073 (NIAID)
 CA 20819 (NCI)
 RR37 (NCRR)
 SOURCE: Clinical and experimental immunology, (1983 Sep) Vol. 53, No. 3, pp. 678-88.
 Journal code: 0057202. ISSN: 0009-9104.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

198311

ENTRY DATE:

Entered STN: 19 Mar 1990

Last Updated on STN: 3 Feb 1997

Entered Medline: 23 Nov 1983

ABSTRACT:

Lymphocyte and neutrophil locomotion were studied in 23 patients with well defined, primary immunodeficiencies. These included eight patients with common variable immune deficiency, three patients with X-linked agammaglobulinaemia, two patients with the Wiskott-Aldrich syndrome, three patients with ***ataxia*** telangiectasia, three patients with immunodeficiency and normal serum immunoglobulin concentrations, one patient with immune deficiency and hyper-IgM syndrome, two patients with Job syndrome and one patient with a granulocyte adherence defect. Random and stimulated lymphocyte and neutrophil migration were evaluated. C5a and casein were used to stimulate lymphocyte migration and C5a and formyl-methionyl-leucyl-phenylalanine (f-MLP) were used to stimulate neutrophil migration. Significantly depressed lymphocyte migration in response to casein and C5a was observed in patients with common variable immune deficiency, patients with immune deficiency and normal immunoglobulin concentration, and patients with Job syndrome. No consistent defect in lymphocyte locomotion was observed in the other patients studied. Neutrophil migration in response to C5a and f-MLP was depressed in Job syndrome, the patient with a granulocyte adherence defect, one of the six patients with common variable immune deficiency and none of the remaining patients. No significant correlation of skin test reactivity and lymphocyte migration was noted, but a correlation between the degree of lymphocyte proliferation in response to phytohaemagglutinin and lymphocyte migration in response to casein was observed. The results presented indicate that aberrations in lymphocyte migration occur in several types of immunodeficiency diseases and that defects in lymphocyte and neutrophil migration can occur simultaneously or totally independent of each other.

CONTROLLED TERM: Check Tags: Female; Male

Adolescent

Adult

Aged

Caseins

Chemotactic Factors

*Chemotaxis, Leukocyte

Child

Child, Preschool

Comparative Study

Complement C5

Complement C5a

Humans

Hypersensitivity, Delayed

*Immunologic Deficiency Syndromes: IM, immunology

Job's Syndrome: IM, immunology

Lymphocyte Activation

Lymphocytes: IM, immunology

Middle Aged

Mitosis

N-Formylmethionine Leucyl-Phenylalanine

Neutrophils: IM, immunology

Phytohemagglutinins: PD, pharmacology

Receptors, Antigen, B-Cell: AN, analysis

Research Support, Non-U.S. Gov't

Research Support, U.S. Gov't, P.H.S.

CAS REGISTRY NO.: 59880-97-6 (N-Formylmethionine Leucyl-Phenylalanine);

80295-54-1 (Complement C5a)

CHEMICAL NAME: 0 (Caseins); 0 (Chemotactic Factors); 0 (Complement C5); 0

(Phytohemagglutinins); 0 (Receptors, Antigen, B-Cell)

L221 ANSWER 10 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006092279 EMBASE

TITLE: Silymarin and silibinin cause G1 and G2-M cell cycle arrest via distinct circuitries in human prostate cancer PC3 cells: A comparison of flavanone silibinin with flavanolignan mixture silymarin.

AUTHOR: Deep G.; Singh R.P.; Agarwal C.; Kroll D.J.; Agarwal R.

CORPORATE SOURCE: Prof. R. Agarwal, Department of Pharmaceutical Sciences, School of Pharmacy, University of Colorado Health Sciences Center, 4200 East Ninth Avenue, Denver, CO 80262, United States. Rajesh.Agarwal@UCHSC.edu

SOURCE: Oncogene, (16 Feb 2006) Vol. 25, No. 7, pp. 1053-1069. .

Refs: 85

ISSN: 0950-9232 E-ISSN: 1476-5594 CODEN: ONCNES

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT:

016	Cancer
028	Urology and Nephrology
030	Pharmacology
037	Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 16 Mar 2006
Last Updated on STN: 16 Mar 2006

ABSTRACT: Here, we assessed and compared the anticancer efficacy and associated mechanisms of silymarin and silibinin in human prostate cancer (PCA) PC3 cells; silymarin is comprised of silibinin and its other stereoisomers, including isosilybin A, isosilybin B, silydianin, silychristin and isosilychristin. Silymarin and silibinin (50-100 µg/ml) inhibited cell proliferation, induced cell death, and caused G1 and G2-M cell cycle arrest in a dose/time-dependent manner. Molecular studies showed that G1 arrest was associated with a decrease in cyclin D1, cyclin D3, cyclin E, cyclin-dependent kinase (CDK) 4, CDK6 and CDK2 protein levels, and CDK2 and CDK4 kinase activity, together with an increase in CDK inhibitors (CDKIs) Kip1/p27 and Cip1/p21. Further, both agents caused cytoplasmic sequestration of cyclin D1 and CDK2, contributing to G1 arrest. The G2-M arrest by silibinin and silymarin was associated with decreased levels of cyclin B1, cyclin A, pCdc2 (Tyr15), Cdc2, and an inhibition of Cdc2 kinase activity. Both agents also decreased the levels of Cdc25B and cell division cycle 25C (Cdc25C) phosphatases with an increased phosphorylation of Cdc25C at Ser216 and its translocation from nucleus to the cytoplasm, which was accompanied by an increased binding with 14-3-3β. Both agents also increased checkpoint kinase (Chk)2 phosphorylation at Thr68 and Ser19 sites, which is known to phosphorylate Cdc25C at Ser216 site. Chk2-specific small interfering RNA largely attenuated the silymarin and silibinin-induced G2-M arrest. An increase in the phosphorylation of histone 2AX and *ataxia telangiectasia* mutated was also observed. These findings indicate that silymarin and silibinin modulate G1 phase cyclins-CDKs-CDKIs for G1 arrest, and the Chk2-Cdc25C-Cdc2/cyclin B1 pathway for G2-M arrest, together with an altered subcellular localization of critical cell cycle regulators. Overall, we observed comparable effects for both silymarin and silibinin at equal concentrations by weight, suggesting that silibinin could be a major cell cycle-inhibitory component in silymarin. However, other silibinin stereoisomers present in silymarin also contribute to its efficacy, and could be of interest for future investigation. .COPYRGT. 2006 Nature Publishing Group All rights reserved.

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Handy 10/617943

11/06/2006

CONTROLLED TERM: Medical Descriptors:

*cell cycle arrest
*antineoplastic activity
*prostate cancer
cell cycle G1 phase
cell cycle G2 phase
cell cycle M phase
cancer cell culture
comparative study
drug mechanism
drug efficacy
stereoisomerism
concentration response
cell proliferation
cell death
molecular biology
protein content
protein determination
enzyme activity
cytoplasm
protein localization
cellular distribution
enzyme inhibition
quantitative analysis
enzyme phosphorylation
intracellular transport
cell nucleus
protein transport
protein protein interaction
cell cycle regulation
drug effect
drug potency
human

male
controlled study
human cell
article
priority journal

CONTROLLED TERM: Drug Descriptors:

**silibinin*: CM, drug comparison
**silibinin*: DV, drug development
**silibinin*: PD, pharmacology
**silylmarin*: CM, drug comparison
**silylmarin*: DV, drug development
**silylmarin*: PD, pharmacology
**flavanoid*: CM, drug comparison
**flavanoid*: DV, drug development
**flavanoid*: PD, pharmacology
*lignan derivative: CM, drug comparison
*lignan derivative: DV, drug development
*lignan derivative: PD, pharmacology
isosilybin A
isosilybin B
 siliidianin
 silicristin
isosilychristin
cyclin D1: EC, endogenous compound
 cyclin D3: EC, endogenous compound
cyclin E: EC, endogenous compound
cyclin dependent kinase 4: EC, endogenous compound

cyclin dependent kinase 6: EC, endogenous compound
 cyclin dependent kinase 2: EC, endogenous compound
 cyclin dependent kinase inhibitor 1B: EC, endogenous compound
 cyclin dependent kinase inhibitor 1: EC, endogenous compound
 cyclin B1: EC, endogenous compound
 cyclin A: EC, endogenous compound
 cell cycle protein: EC, endogenous compound
 cyclin dependent kinase 1: EC, endogenous compound
 phosphatase: EC, endogenous compound
 protein 14 3 3: EC, endogenous compound
 checkpoint kinase 2: EC, endogenous compound
 small interfering RNA
 histone H2AX: EC, endogenous compound
 ATM protein: EC, endogenous compound
 unclassified drug

CAS REGISTRY NO.: (silibinin) 22888-70-6; (silidianin) 29782-68-1;
 (silicristin) 33889-69-9; (cyclin dependent kinase 4)
 147014-97-9; (cyclin dependent kinase 2) 141349-86-2;
 (phosphatase) 9013-05-2; (protein 14 3 3) 136047-16-0;
 (checkpoint kinase 2) 244634-79-5

COMPANY NAME: Sigma Aldrich (United States)

L221 ANSWER 11 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005206506 EMBASE

TITLE: Farnesyltransferase inhibitors induce DNA damage via reactive oxygen species in human cancer cells.

AUTHOR: Pan J.; She M.; Xu Z.-X.; Sun L.; Yeung S.-C.J.

CORPORATE SOURCE: S.-C.J. Yeung, Dept. of Endocr. Neoplasia/Horm. D., Univ. Texas M.D. Anderson Cancer C., 1515 Holcombe Boulevard, Houston, TX 77030, United States. syeung@mdanderson.org

SOURCE: Cancer Research, (1 May 2005) Vol. 65, No. 9, pp. 3671-3681. .

Refs: 51

ISSN: 0008-5472 CODEN: CNREA8

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 2 Jun 2005

Last Updated on STN: 2 Jun 2005

ABSTRACT: Farnesyltransferase inhibitors (FTIs) possess antitumor activity. Based on recent findings, we hypothesized that FTIs induce reactive oxygen species (ROS) that damage DNA, leading to DNA damage responses. To test this hypothesis, we investigated the effects of FTIs on the generation of ROS, DNA double-strand breaks (DSB), DNA damage responses, and RhoB, and the effects of quenching ROS on these FTI effects. We evaluated four FTIs in human cancer cell lines of different tissue origins. We found that FTIs induced ROS and DSBs. Suppressing expression of the β-subunit of farnesyltransferase with siRNA did not induce ROS, but slightly attenuated the ROS induced by FTIs. N-acetyl-L-cysteine (NAC), but not caspase inhibitors, blocked FTI-induced DSBs, suggesting that the DSBs were caused by ROS and did not result from apoptosis. The DSBs led to DNA damage responses. H2AX became phosphorylated and formed nuclear foci. The DNA-damage-sensing molecules involved were probably *ataxia-telangiectasia* mutated protein (ATM) and DNA-dependent protein kinase (DNA-PK) but not ATM- and Rad3-related protein

(ATR). Key components of the homologous recombination and nonhomologous end-joining repair pathways (DNA-PK, BRCA1, and NBS1) underwent phosphorylation and formed nuclear foci. RhoB, a mediator of the antineoplastic effect of FTIs and a protein inducible by DNA damage, was increased by FTIs. This increase was blocked by NAC. We concluded that FTIs induced oxidative DNA damage by inducing ROS and initiated DNA damage responses, including RhoB induction, and there was a complex relationship among FTIs, farnesyltransferase, ROS, and RhoB. Our data also imply that inhibitors of DNA repair may accentuate the clinical efficacy of FTIs.

CONTROLLED TERM: Medical Descriptors:
*DNA damage
*cancer cell
oxidative stress
DNA strand breakage
evaluation
homologous recombination
antineoplastic activity
protein induction
ataxia telangiectasia
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*protein farnesyltransferase inhibitor: PD, pharmacology
*DNA: EC, endogenous compound
*reactive oxygen metabolite: EC, endogenous compound
manumycin: PD, pharmacology
wortmannin: PD, pharmacology
caffeine: PD, pharmacology
doxycycline: PD, pharmacology
2 [[2 [[2 [(2 amino 3 mercaptopropyl)amino] 3 methylpentyl]oxy] 1 oxo 3 phenylpropyl]amino] 4 (methylsulfonyl)butanoic acid isopropyl ester: PD, pharmacology
n [[5 [(2 amino 3 mercaptopropyl)amino] [1,1' biphenyl] 2 yl]carbonyl]methionine: PD, pharmacology
acetylcysteine: PD, pharmacology
deferoxamine: PD, pharmacology
ATM protein: EC, endogenous compound
DNA dependent protein kinase: EC, endogenous compound
checkpoint kinase Rad3: EC, endogenous compound
BRCA1 protein: EC, endogenous compound
protein nbs1: EC, endogenous compound
RhoB guanine nucleotide binding protein: EC, endogenous compound
unclassified drug
CAS REGISTRY NO.: (DNA) 9007-49-2; (manumycin) 52665-74-4; (wortmannin) 19545-26-7; (caffeine) 30388-07-9, 58-08-2; (doxycycline) 10592-13-9, 17086-28-1, 564-25-0; (2 [[2 [[2 [(2 amino 3 mercaptopropyl)amino] 3 methylpentyl]oxy] 1 oxo 3 phenylpropyl]amino] 4 (methylsulfonyl)butanoic acid isopropyl ester) 160141-09-3; (n [[5 [(2 amino 3 mercaptopropyl)amino] [1,1' biphenyl] 2 yl]carbonyl]methionine) 170006-72-1; (acetylcysteine) 616-91-1; (**deferoxamine**) 70-51-9
CHEMICAL NAME: L 744832; Fti 276

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ACCESSION NUMBER: 2005396236 EMBASE

TITLE: The DNA damage pathway regulates innate immune system ligands of the NKG2D receptor.

AUTHOR: Gasser S.; Orsulic S.; Brown E.J.; Raulet D.H.

CORPORATE SOURCE: D.H. Raulet, Department of Molecular and Cell Biology, Cancer Research Laboratory, University of California, Berkeley, CA 94720-3200, United States.
raulet@uclink.berkeley.edu

SOURCE: Nature, (25 Aug 2005) Vol. 436, No. 7054, pp. 1186-1190.

Refs: 27

ISSN: 0028-0836 CODEN: NATUAS

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 29 Sep 2005
Last Updated on STN: 29 Sep 2005

ABSTRACT: Some stimulatory receptors of the innate immune system, such as the NKG2D receptor (also called KLRK1) expressed by natural killer cells and activated CD8(+)T cells, recognize self-molecules that are upregulated in diseased cells by poorly understood mechanisms. Here we show that mouse and human NKG2D ligands are upregulated in non-tumour cell lines by genotoxic stress and stalled DNA replication, conditions known to activate a major DNA damage checkpoint pathway initiated by ATM (ataxia-telangiectasia, mutated) or ATR (ATM- and Rad3-related) protein kinases. Ligand upregulation was prevented by pharmacological or genetic inhibition of ATR, ATM or Chk1 (a downstream transducer kinase in the pathway). Furthermore, constitutive ligand expression by a tumour cell line was inhibited by targeting short interfering RNA to ATM, suggesting that ligand expression in established tumour cells, which often harbour genomic irregularities, may be due to chronic activation of the DNA damage response pathway. Thus, the DNA damage response, previously shown to arrest the cell cycle and enhance DNA repair functions, or to trigger apoptosis, may also participate in alerting the immune system to the presence of potentially dangerous cells.

CONTROLLED TERM: Medical Descriptors:
*DNA damage
*immune system
immunoregulation
regulatory mechanism
natural killer cell
gene expression
T lymphocyte activation
genotoxicity
stress
DNA replication
enzyme inhibition
tumor cell line
gene targeting
mitosis inhibition
DNA repair
apoptosis
nonhuman
mouse
controlled study
animal cell

article
priority journal
Drug Descriptors:
*natural killer cell receptor NKG2D: EC, endogenous compound
ligand
CD8 antigen: EC, endogenous compound
ATM protein
ATR protein
protein kinase
checkpoint kinase 1
small interfering RNA
CAS REGISTRY NO.: (protein kinase) 9026-43-1

L221 ANSWER 13 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005357623 EMBASE
TITLE: ATM Polymorphism and hereditary nonpolyposis colorectal cancer (HNPCC) age of onset (United States).
AUTHOR: Jones J.S.; Gu X.; Lynch P.M.; Rodriguez-Bigas M.; Amos C.I.; Frazier M.L.
CORPORATE SOURCE: Dr. M.L. Frazier, Department of Epidemiology, University of Texas, M.D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, United States.
mlfrazier@mdanderson.org
SOURCE: Cancer Causes and Control, (2005) Vol. 16, No. 6, pp. 749-753.
Refs: 16
ISSN: 0957-5243 CODEN: CCCNEN
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
022 Human Genetics
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Sep 2005
Last Updated on STN: 9 Sep 2005

ABSTRACT: Objective: We examined a G-to-A single nucleotide polymorphism of the ATM gene, to determine if it influences hereditary non-polyposis colorectal cancer (HNPCC) age of onset. HNPCC is caused by mutations in mismatch repair genes, especially hMLH1 and hMSH2. ATM germline mutations have been associated with breast and digestive cancers. In a smaller European study, the G-to-A polymorphism was associated with an increased risk of developing an HNPCC-related cancer within HNPCC families. Materials and methods: We genotyped 109 mismatch repair gene (MMR) mutation carriers from 53 HNPCC families for the ATM polymorphism using PCR and single strand conformational polymorphism (SSCP) analysis. We tested the association between the ATM genotypes and HNPCC age of onset by survival analysis. Results: The ATM polymorphism did not significantly modify HNPCC age of onset, nor overall risk, in our population. Conclusions: Although a modifier effect was not seen in our study, future studies that examine the polymorphism in combination with other genetic and environmental factors may elucidate an association. Revealing such associations in MMR mutation carriers may improve risk estimates and help to identify individuals who are genetically susceptible to developing HNPCC at an earlier age. .COPYRGT. Springer 2005.

CONTROLLED TERM: Medical Descriptors:
*genetic polymorphism
*colorectal cancer

onset age
United States
genotype
mismatch repair
gene mutation
family history
polymerase chain reaction
single strand conformation polymorphism
cancer survival
statistical significance
cancer risk
statistical model
genotype environment interaction
risk assessment
genetic susceptibility
 ataxia telangiectasia
human
male
female
major clinical study
controlled study
aged
adult
article
priority journal
Drug Descriptors:
borinic acid derivative
 edetic acid

CAS REGISTRY NO.: (edetic acid) 150-43-6, 60-00-4

L221 ANSWER 14 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2004397797 EMBASE
TITLE: The genetics of hypogammaglobulinemia.
AUTHOR: Grimbacher B.; Schaffer A.A.; Peter H.-H.
CORPORATE SOURCE: Dr. B. Grimbacher, Div. of Rheumatol./Clin. Immunology,
Medical School, University of Freiburg, Hugstetterstrasse
55, 79106 Freiburg, Germany. grimbacher@medizin.ukl.uni-freiburg.de
SOURCE: Current Allergy and Asthma Reports, (2004) Vol. 4, No. 5,
pp. 349-358. .
Refs: 90
ISSN: 1529-7322 CODEN: CAARC
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
 022 Human Genetics
 026 Immunology, Serology and Transplantation
 037 Drug Literature Index
 038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 2004
 Last Updated on STN: 30 Sep 2004
ABSTRACT: Etiologies for human hypogammaglobulinemias are diverse and include genetic and nongenetic causes. Although recent reviews focus on the complex genetics of common variable immunodeficiency, in this review, we survey different causes of hypogammaglobulinemias and discuss possible mechanisms.
Copyright .COPYRGT. 2004 by Current Science Inc.

CONTROLLED TERM: Medical Descriptors:
*molecular genetics
*hypogammaglobulinemia: DT, drug therapy
*hypogammaglobulinemia: ET, etiology
*hypogammaglobulinemia: SI, side effect
genetic susceptibility
disease classification
humoral immune deficiency: DT, drug therapy
humoral immune deficiency: ET, etiology
humoral immune deficiency: SI, side effect
common variable immunodeficiency: ET, etiology
immunoglobulin G deficiency: DT, drug therapy
immunoglobulin G deficiency: ET, etiology
immunoglobulin G deficiency: SI, side effect
immunoglobulin A deficiency: ET, etiology
immunoglobulin A deficiency: SI, side effect
infant disease: ET, etiology
linkage analysis
Wiskott Aldrich syndrome: DT, drug therapy
 ataxia telangiectasia: DT, drug therapy
DiGeorge syndrome: DT, drug therapy
human
review
Drug Descriptors:
immunoglobulin G: EC, endogenous compound
immunoglobulin A: EC, endogenous compound
cytotoxic agent: AE, adverse drug reaction
B lymphocyte antibody: AE, adverse drug reaction
gold: AE, adverse drug reaction
corticosteroid: AE, adverse drug reaction
salazosulfapyridine: AE, adverse drug reaction
chloroquine: AE, adverse drug reaction
 penicillamine: AE, adverse drug reaction
hydantoin: AE, adverse drug reaction
carbamazepine: AE, adverse drug reaction
zonisamide: AE, adverse drug reaction
valproic acid: AE, adverse drug reaction
captopril: AE, adverse drug reaction
fenclofenac: AE, adverse drug reaction
polysaccharide vaccine: DT, drug therapy
(immunoglobulin G) 97794-27-9; (gold) 7440-57-5;
(salazosulfapyridine) 599-79-1; (chloroquine) 132-73-0,
3545-67-3, 50-63-5, 54-05-7; (*penicillamine*)
2219-30-9, 52-67-5; (hydantoin) 461-72-3;
(carbamazepine) 298-46-4, 8047-84-5; (zonisamide)
68291-97-4; (valproic acid) 1069-66-5, 99-66-1; (captopril)
62571-86-2; (fenclofenac) 34645-84-6

CAS REGISTRY NO.: L221 ANSWER 15 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 81251649 EMBASE
DOCUMENT NUMBER: 1981251649
TITLE: The *ataxia telangiectasia* clastogenic factor is a low molecular weight peptide.
AUTHOR: Shaham M.; Becker Y.
CORPORATE SOURCE: Dept. Hum. Genet. Molec. Virol., Hadassah-Hebrew Univ. Med. Cent., Jerusalem, Israel
SOURCE: Human Genetics, (1981) Vol. 58, No. 4, pp. 422-424.
COUNTRY: Germany

DOCUMENT TYPE: Journal
 FILE SEGMENT: 022 Human Genetics
 008 Neurology and Neurosurgery
 037 Drug Literature Index

LANGUAGE: English
 ENTRY DATE: Entered STN: 9 Dec 1991
 Last Updated on STN: 9 Dec 1991

ABSTRACT: The clastogenic factor in the plasma of **ataxia telangiectasia** (AT) patients and in conditioned medium from AT skin fibroblast cultures is a peptide with a molecular weight in the range of 500 to 1000. No clastogenic activity could be demonstrated in extracts of cultured AT fibroblasts.

CONTROLLED TERM: Medical Descriptors:
 ***ataxia telangiectasia**
 *chromosome breakage
 *clastogenesis
 *fibroblast
 cell culture
 fibroblast culture
 molecular weight
 peptide analysis
 serum
 in vitro study
 Drug Descriptors:
 *peptide
 *phytohemagglutinin
 edetic acid
 trypsin

CAS REGISTRY NO.: (phytohemagglutinin) 9008-97-3; (**edetic acid**) 150-43-6, 60-00-4; (trypsin)
 9002-07-7

COMPANY NAME: Wellcome103 (United Kingdom); 101; 111; 912; 923; 924

L221 ANSWER 16 OF 24 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
 ACCESSION NUMBER: 81125089 EMBASE
 DOCUMENT NUMBER: 1981125089
 TITLE: Effect of the flavonoid (+) cyanidanol-3 on procollagen biosynthesis and transport in normal and **ataxia telangiectasia** cultured skin fibroblasts.
 AUTHOR: Becker Y.; Stevely W.; Hamburger Y.; et al.
 CORPORATE SOURCE: Dept. Molec. Virol., Hebrew Univ. Hadassah Med. Sch., Jerusalem, Israel
 SOURCE: Connective Tissue Research, (1981) Vol. 8, No. 2, pp. 77-84.
 CODEN: CVTRBC
 COUNTRY: United States
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 029 Clinical Biochemistry
 022 Human Genetics
 008 Neurology and Neurosurgery
 037 Drug Literature Index

LANGUAGE: English
 ENTRY DATE: Entered STN: 9 Dec 1991
 Last Updated on STN: 9 Dec 1991

ABSTRACT: The synthesis and secretion of procollagen into the medium of cultures of human skin fibroblasts from normal individuals and from patients with the genetic disorder, **ataxia telangiectasia**, are markedly inhibited by the flavonoid (+)cyanidanol-3. Those proteins which were

secreted into the medium in the presence of cyanidanol were resistant to collagenase treatment (noncollagenous proteins). Polyacrylamide gel electrophoresis revealed the presence of only one noncollagenous protein of 66,000 daltons in the medium of cyanidanol-treated cells as compared with the nine other polypeptides found in the medium of untreated cells.

CONTROLLED TERM: Medical Descriptors:
*ataxia telangiectasia
*fibroblast
cell culture
cysteine s 35
fibroblast culture
skin
central nervous system
in vitro study
human cell
peripheral vascular system
reticuloendothelial system
heredity
major clinical study
Drug Descriptors:
*2,2' bipyridine
*aminoacetonitrile
*3 aminopropionitrile
*catechin
*penicillamine
*procollagen
radioisotope
CAS REGISTRY NO.: (2,2' bipyridine) 366-18-7; (aminoacetonitrile) 151-63-3,
540-61-4; (3 aminopropionitrile) 151-18-8; (catechin)
13392-26-2, 154-23-4; (penicillamine) 2219-30-9,
52-67-5
COMPANY NAME: Zyma (Switzerland); Aldrich (United States)

L221 ANSWER 17 OF 24 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2006:182247 BIOSIS
DOCUMENT NUMBER: PREV200600184359
TITLE: CML progenitor cells have chromosomal instability and
display increased DNA damage at DNA fragile sites.
AUTHOR(S): Dierov, Jamil K. [Reprint Author]; Schoppay, David W.;
Carroll, Martin
CORPORATE SOURCE: Univ Penn, Philadelphia, PA 19104 USA
SOURCE: Blood, (NOV 16 2005) Vol. 106, No. 11, Part 1, pp. 563A.
Meeting Info.: 47th Annual Meeting of the
American-Society-of-Hematology. Atlanta, GA, USA. December
10 -13, 2005. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Mar 2006
Last Updated on STN: 15 Mar 2006

ABSTRACT: Chronic myelogenous leukemia (CML) is a two stage disease which progresses to blast crisis over a period of 3-5 years in untreated patients. The BCR/ABL oncogene induces the hyperproliferation associated with chronic phase CML but whether BCR/ABL induces chromosomal instability leading to blast crisis has been controversial. We have previously demonstrated that BCR/ABL delays the repair of DNA double strand breaks and increases chromosomal instability in a murine cell line. Furthermore, we have demonstrated in cell

lines that *BCR/ABL* disrupts the function of the DNA damage sensing protein, *ataxia telangiectasia* and *rad 3 related (ATR)*. One of the functions of ATR is to maintain the stability of DNA fragile sites, late replicating sites in the chromosome that are frequently involved in translocations. To determine if *BCR/ABL* affects the stability of DNA fragile sites in Bat F3 cells that do or do not express *BCR/ABL*, cells were incubated in low dose aphidicolin for 24 hours to induce fragile site breakage. *BCR/ABL* expressing cells, but not control cells, demonstrated fragile site damage consistent with a disruption of ATR function in *BCR/ABL* expressing cells. In order to determine if primary patient cells display a genomic instability phenotype, we have analyzed the response to DNA damage in CD34+ cells from normal volunteers and from CIVIL patients seen at the University of Pennsylvania Cancer Center. We first examined the DNA repair response by treating cells for two hours with etoposide. Both normal cells and CML progenitor cells demonstrate DNA double strand breaks as measured by the comet assay, a quantitative assay for DNA double strand breaks. However, in Ph+ cells from the patient sample there was a delay in the repair of DNA double strand breaks as indicated by a significant increase in the olive tail moment at 2 hours and 24 hours after treatment with etoposide. In addition, we analyzed the effect of a two hour exposure to etoposide on chromosome stability as measured by spectral karyotyping (SKY). Normal CD34+ cells and CD34+ cells from patients were treated with etoposide and then allowed to recover for 48 hours before analysis of metaphase spreads. Normal cells demonstrated no spontaneous DNA damage and, after etoposide treatment and repair, demonstrated only modest levels of DNA damage (2 translocations and 5 numerical alterations per 14 metaphases analyzed). In contrast, Ph+ cells demonstrated spontaneous DNA damage in these cell conditions. Furthermore, after etoposide treatment Ph+ cells demonstrated high levels of DNA damage with 9 translocations and 12 numerical alterations in 13 metaphases. These results suggest that Ph+ progenitor cells from patients with CML demonstrate chromosomal instability and suggest a mechanism for progression from CML chronic phase to blast crisis. Full analysis of additional patient samples will be presented. Taken together, we propose that *BCR/ABL* disrupts ATR function in cell lines and primary cells leading to an increase in chromosomal instability that leads to CML blast crisis.

CONCEPT CODE: General biology - Symposia, transactions and proceedings
 00520
 Cytology - Animal 02506
 Cytology - Human 02508
 Genetics - General 03502
 Genetics - Animal 03506
 Genetics - Human 03508
 Biochemistry studies - General 10060
 Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
 Pathology - Therapy 12512
 Blood - Blood and lymph studies 15002
 Blood - Blood cell studies 15004
 Blood - Blood, lymphatic and reticuloendothelial pathologies 15006
 Pharmacology - Clinical pharmacology 22005
 Pharmacology - Blood and hematopoietic agents 22008
 Neoplasms - Immunology 24003
 Neoplasms - Pathology, clinical aspects and systemic effects 24004
 Neoplasms - Therapeutic agents and therapy 24008
 Neoplasms - Blood and reticuloendothelial neoplasms 24010
 Immunology - General and methods 34502
 Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS: Major Concepts

11/06/2006

Handy 10/617943

11/06/2006

INDEX TERMS: Molecular Genetics (Biochemistry and Molecular Biophysics); Oncology (Human Medicine, Medical Sciences); Hematology (Human Medicine, Medical Sciences)

INDEX TERMS: Parts, Structures, & Systems of Organisms

progenitor cell: blood and lymphatics; CD34-positive cell: immune system, blood and lymphatics; chromosome

INDEX TERMS: Diseases

chronic myelogenous leukemia: neoplastic disease, blood and lymphatic disease, drug therapy, CML

INDEX TERMS: Chemicals & Biochemicals

DNA; etoposide: antineoplastic-drug, hematologic-drug

INDEX TERMS: Methods & Equipment

spectral karyotyping: laboratory techniques, genetic techniques

ORGANISM: Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

human (common)

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGANISM: Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

Ba/F3 cell line (cell_line): murine pro-B cell

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER: 33419-42-0 (etoposide)

GENE NAME: human BCR/ABL gene (Hominidae)

L221 ANSWER 18 OF 24 USPATFULL on STN

ACCESSION NUMBER: 2006:9627 USPATFULL

TITLE: Shiga toxin B-subunit as a vector for tumor diagnosis and drug delivery to Gb3 expressing tumors

INVENTOR(S): Johannes, Ludger, Courbevoie, FRANCE

Grierson, David, Versailles, FRANCE

Robine, Sylvie, Vanves, FRANCE

Florent, Jean-Claude, Gif-Sur-Yvette, FRANCE

Maillard, Philippe, Saint-Cyr-L'Ecole, FRANCE

Roger, Jacky, Villecresnes, FRANCE

PATENT ASSIGNEE(S): INSTITUT CURIE, Paris Cedex 05, FRANCE (non-U.S. corporation)

CENTRE NATIONAL DE LA RECHERCHE SCIENTIFIQUE, Paris Cedex, FRANCE (non-U.S. corporation)

INSTITUT NATIONAL DE LA SANTE ET DE LA RECHERCHE MEDICALE, Paris Cedex 13, FRANCE (non-U.S. corporation)

UNIVERSITE PIERRE ET MARIE CURIE (PARIS VI), Paris, FRANCE (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006008475	A1	20060112

APPLICATION INFO.: US 2005-46786 A1 20050201 (11)
RELATED APPLN. INFO.: Continuation of Ser. No. WO 2003-EP9308, filed on 31
Jul 2003, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2002-291962	20020802
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	BIRCH STEWART KOLASCH & BIRCH, PO BOX 747, FALLS CHURCH, VA, 22040-0747, US	
NUMBER OF CLAIMS:	18	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	16 Drawing Page(s)	
LINE COUNT:	1412	
CAS INDEXING IS AVAILABLE FOR THIS PATENT		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
The citation relates to new compound

AB The invention relates to new compounds for cancer therapy or diagnosis and to the use of a non-toxic B subunit of Shiga toxin mutant as a vector for diagnostic products or drugs in over-expressing Gb3 receptor cells, such compounds having the following formula: STxB-Z(n)-Cys-Y(m)-T wherein --STxB is the Shiga Toxin B subunit or a functional equivalent thereof, --Z(n) wherein n is 0 or 1, Z is an amino-acid residue devoid of sulphydryl groups, or is a polypeptide, --T is a molecule linked by a covalent bound to the S part of Cys, selected from: agents for in vivo diagnosis, cytotoxic agents, prodrugs, or enzymes for the conversion of a prodrug to a drug, --Y(m) wherein m is 0 or 1, Y is a linker between T and Cys, which is either cleavable or not cleavable for the release of T after the internalization of the hybrid compound into cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

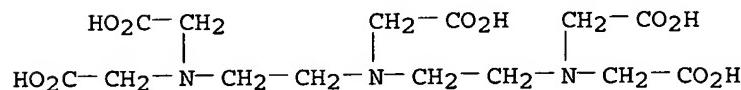
DETD Babilon, R. W., K. J. Soprano, and E. E. Henderson. 1985. Hypersensitivity and reduced inhibition of DNA synthesis in *ataxia telangiectasia* lymphoblasts treated with low levels of neocarzinostatin. Mutat. Res. 146:79-87.

DETD Shiloh, Y., E. Tabor, and Y. Becker. 1982. Cellular hypersensitivity to neocarzinostatin in *ataxia-telangiectasia* skin fibroblasts. *Cancer Res.* 42:2247-2249.

IT 67-43-6D, Diethylenetriaminepentaacetic acid, complex with gadolinium and ethoxybenzyl 7440-54-2D, Gadolinium, complex with acetic acid derivs. 7440-54-2D, Gadolinium, polymer complexes 83678-67-5, Gd-DOTA 651740-21-5
(Shiga toxin B-subunit as a vector for tumor diagnosis and drug delivery to Gb3-expressing tumors)

IT 67-43-6D, Diethylenetriaminepentaacetic acid, complex with gadolinium and ethoxybenzyl
(Shiga toxin B-subunit as a vector for tumor diagnosis and drug delivery to Gb3-expressing tumors)

RN 67-43-6 USPATFULL
CN Glycine, N,N-bis[2-[bis(carboxymethyl)amino]ethyl]- (7CI, 8CI, 9CI) (CA
INDEX NAME)



L221 ANSWER 19 OF 24 USPATFULL on STN
ACCESSION NUMBER: 2005:292956 USPATFULL

TITLE: Method for determination and quantification of radiation or genotoxin exposure
 INVENTOR(S): D'Andrea, Alan D., Winchester, MA, UNITED STATES
 PATENT ASSIGNEE(S): Dana Farber Cancer Center (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005255502	A1	20051117
APPLICATION INFO.:	US 2005-46346	A1	20050128 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-540380P	20040130 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PALMER & DODGE, LLP, KATHLEEN M. WILLIAMS, 111 HUNTINGTON AVENUE, BOSTON, MA, 02199, US	
NUMBER OF CLAIMS:	68	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	14 Drawing Page(s)	
LINE COUNT:	2700	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention discloses methods for detecting exposure of a living subject to genotoxic agents, testing sensitivity to a genotoxic agent, and determining DNA damage caused by exposure to an agent, comprising detecting the presence of FANCD2-containing foci from a sample collected from said subject. The presence of concentrated foci is indicative of DNA damage, and the degree of foci formation is correlated with degree of exposure. Diagnostic reagents contain a ligand that binds to human FANCD2 associated with a detectable label. Kits for detecting DNA damage in a biological sample contain such diagnostic reagents and signal detection components. The invention further discloses methods for identifying agents which modulate the ability of FANCD2-containing foci to form. Among other things, such agents are potentially useful chemosensitizing agents or may confer protection against damage caused by genotoxic agents.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Kim, S. T., Lane, W. S., Kastan, M. B., and D'Andrea, A. D. (2002). Convergence of the Fanconi anemia and **ataxia telangiectasia** signaling pathways. *Cell* 109, 459-472.

Wu, X., Petrini, J. H., Heine, W. F., Weaver, D. T., Livingston, D. M., . . .

IT 70-51-9, Desferrioxamine

(FA/BRCA pathway agonist; antibodies to monoubiquitinated FANCD2 protein and method for determination and quantification of radiation or genotoxin exposure)

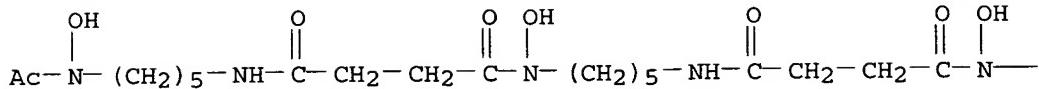
IT 70-51-9, Desferrioxamine

(FA/BRCA pathway agonist; antibodies to monoubiquitinated FANCD2 protein and method for determination and quantification of radiation or genotoxin exposure)

RN 70-51-9 USPATFULL

CN Butanediamide, N'-[5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl]-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B

 $\text{---} - (\text{CH}_2)_5 - \text{NH}_2$

L221 ANSWER 20 OF 24 USPATFULL on STN

ACCESSION NUMBER: 2005:286471 USPATFULL

TITLE: Methods of treating ankylosing spondylitis using anti-TNF antibodies and peptides of human tumor necrosis factor

INVENTOR(S): Le, Junming, Forest Hills, NY, UNITED STATES
Vilcek, Jan T., Manhattan, NY, UNITED STATES
Daddona, Peter E., Menlo Park, CA, UNITED STATES
Gharyeb, John, Downingtown, PA, UNITED STATES
Knight, David M., Berwyn, PA, UNITED STATES
Siegel, Scott A., Ringoes, NJ, UNITED STATES
Shealy, David J., Downingtown, PA, UNITED STATES
Centocor, Inc., Malvern, PA, UNITED STATES (U.S. corporation)
New York University, New York, NY, UNITED STATES (U.S. corporation)

NUMBER	KIND	DATE
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US 2005249735	A1	20051110
US 2004-10954	A1	20041213 (11)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2003-637759, filed on 8 Aug 2003, PENDING Continuation-in-part of Ser. No. US 2001-927703, filed on 10 Aug 2001, PENDING Continuation of Ser. No. US 2001-756398, filed on 8 Jan 2001, GRANTED, Pat. No. US 6835823 Continuation-in-part of Ser. No. US 2001-920137, filed on 1 Aug 2001, PENDING
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NUMBER	DATE
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PRIORITY INFORMATION:	US 2000-223360P 20000807 (60)
	US 2000-236826P 20000929 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US

NUMBER OF CLAIMS: 34

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 37 Drawing Page(s)

LINE COUNT: 7263

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

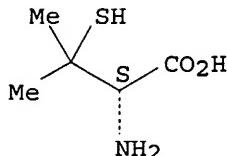
AB Anti-TNF antibodies, fragments and regions thereof which are specific

for human tumor necrosis factor- α (TNF α) and are useful in vivo diagnosis and therapy of a number of TNF α -mediated pathologies and conditions, including ankylosing spondylitis, as well as polynucleotides coding for murine and chimeric antibodies, methods of producing the antibody, methods of use of the anti-TNF antibody, or fragment, region or derivative thereof, in immunoassays and immunotherapeutic approaches are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

- DETD . . . ataxia, Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and Machado-Joseph); and systemic disorders (Refsum's disease, abetalipoproteinemia, **ataxia, telangiectasia**, and mitochondrial multi-system disorder); demyelinating core disorders, such as multiple sclerosis, acute transverse myelitis; disorders of the motor unit, such. . .
- DETD . . . spinal ataxia, Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and Machado-Joseph); systemic disorders (Refsum's disease, abetalipoproteinemia, **ataxia, telangiectasia**, and mitochondrial multi-system disorder); demyelinating core disorders, such as multiple sclerosis, acute transverse myelitis; and disorders of the motor unit'. . .
- IT 50-07-7, Mitomycin C 50-78-2, Aspirin 51-43-4, Epinephrine 52-28-8, Codeine phosphate 52-67-5, Penicillamine 53-86-1, Indomethacin 54-05-7, Chloroquine 59-05-2, Methotrexate 89-57-6, Asacol 103-90-2, Paracetamol 118-42-3, Hydroxychloroquine 321-64-2, Tacrine 446-86-6, Azathioprine 469-62-5, Dextropropoxyphene 5003-48-5, Benorylate 9002-72-6, Growth hormone 11096-26-7, Erythropoietin 12244-57-4, Myocrisin 15307-86-5, Diclofenac 15687-27-1, Ibuprofen 16110-51-3, Cromolyn 22204-53-1, Naprosyn 23214-92-8, Doxorubicin 28109-92-4, Methylxanthine 34031-32-8, Auranofin 36330-85-5, Fenbufen 41340-25-4, Etodolac 120014-06-4, Donepezil 121181-53-1, Filgrastim 123774-72-1, Sargramostim 143831-71-4, Dornase alfa
(methods of treating ankylosing spondylitis using anti-tumor necrosis factor antibodies and peptides of human tumor necrosis factor)
- IT 52-67-5, Penicillamine
(methods of treating ankylosing spondylitis using anti-tumor necrosis factor antibodies and peptides of human tumor necrosis factor)
- RN 52-67-5 USPATFULL
- CN D-Valine, 3-mercaptop- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L221 ANSWER 21 OF 24 USPATFULL on STN
 ACCESSION NUMBER: 2005:144174 USPATFULL
 TITLE: COMPOSITIONS AND METHODS FOR TREATING CELLS HAVING DOUBLE MINUTE DNA
 INVENTOR(S): WAHL, GEOFFREY M., SAN DIEGO, CA, UNITED STATES
 SHIMIZU, NORIAKI, HIROSHIMA, JAPAN

KANDA, TERU, LA JOLLA, CA, UNITED STATES
 SHEPARD, H. MICHAEL, RANCHO SANTA FE, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005123909	A1	20050609
	US 6946259	B2	20050920
APPLICATION INFO.:	US 1999-229229	A1	19990112 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-71146P	19980112 (60)
	US 1998-77644P	19980311 (60)

DOCUMENT TYPE:

Utility

FILE SEGMENT:

APPLICATION

LEGAL REPRESENTATIVE: MCDERMOTT, WILL & EMERY, 4370 LA JOLLA VILLAGE DRIVE,
SUITE 700, SAN DIEGO, CA, 92122, US

NUMBER OF CLAIMS: 23

EXEMPLARY CLAIM: 1-32

NUMBER OF DRAWINGS: 14 Drawing Page(s)

LINE COUNT: 1977

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides methods by which test substances can be screened for their ability to inhibit, enhance or eliminate double minute (DM) or extrachromosomal DNA by micronucleation in cells. This invention also provides a method for inducing maturation or death of a cell having the capacity to generate micronuclei. It also provides a method of treating a disease in a subject, the cells correlated with the disease having DM and extrachromosomal DNA as well as the capacity to generate micronuclei to capture them. Further provided is a method of detecting chromosomal and extrachromosomal DNA in a cell.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . B. Vogelstein and A. J. Fonace. 1992. "A mammalian cell cycle checkpoint pathway utilizing p53 and GADD45 is defective in ataxia-telangiectasia" Cell. 7:587-97.

Kuerbitz, S. J., B. S. Plunkett, W. V. Walsh and M. B. Kastan. 1992.
"Wild-type p53 is . . .

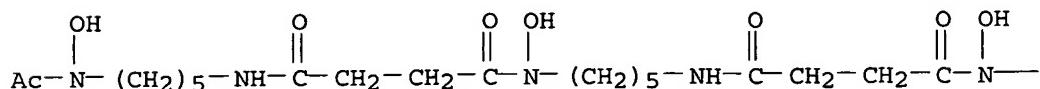
IT 67-68-5, DMSO, biological studies 70-51-9, Deferoxamine
91-64-5, Coumarin 98-92-0, Nicotinamide 1455-77-2, Guanazole
38966-21-1, Aphidicolin 51321-79-0, PALA
(compns. and methods for identifying therapeutic agents and for
treating cells having double minute DNA)

IT 70-51-9, Deferoxamine
(compns. and methods for identifying therapeutic agents and for
treating cells having double minute DNA)

RN 70-51-9 USPATFULL

CN Butanediamide, N'-(5-[[4-[[5-(acetylhydroxyamino)pentyl]amino]-1,4-dioxobutyl]hydroxyamino]pentyl)-N-(5-aminopentyl)-N-hydroxy- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 1-B

 $\text{---} (\text{CH}_2)_5-\text{NH}_2$

L221 ANSWER 22 OF 24 USPATFULL on STN
ACCESSION NUMBER: 2003:161945 USPATFULL
TITLE: Diagnosis and management of infection caused by chlamydia
INVENTOR(S): Mitchell, William M., Nashville, TN, United States
Stratton, Charles W., Nashville, TN, United States
PATENT ASSIGNEE(S): Vanderbilt University, Nashville, TN, United States
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6579854	B1	20030617
APPLICATION INFO.:	US 1998-73661		19980506 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1998-25174, filed on 18 Feb 1998 Continuation-in-part of Ser. No. US 1998-25521, filed on 18 Feb 1998, now abandoned Continuation-in-part of Ser. No. US 1998-25176, filed on 18 Feb 1998, now patented, Pat. No. US 6258532 Continuation-in-part of Ser. No. US 1997-911593, filed on 14 Aug 1997, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-45689P	19970506 (60)
	US 1997-45739P	19970506 (60)
	US 1997-45779P	19970506 (60)
	US 1997-45780P	19970506 (60)
	US 1997-45787P	19970506 (60)
	US 1996-23921P	19960814 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Weddington, Kevin E.
LEGAL REPRESENTATIVE: Clark & Elbing LLP
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 4353

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a unique approach for the diagnosis and management of infections by Chlamydia species, particularly C. pneumoniae. The invention is based, in part, upon the discovery that a combination of agents directed toward the various stages of the chlamydial life cycle is effective in substantially reducing infection. Products comprising combination of antichlamydial agents, novel compositions and pharmaceutical packs are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and Machado Joseph); and systemic disorders (Refsum's disease, abetalipoproteinemia, ataxia, telangiectasia, and mitochondrial multi-system disorder); demyelinating core disorders, such

as multiple sclerosis, acute transverse myelitis; disorders of the motor unit, such.

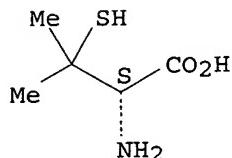
IT 52-67-5, Penicillamine 54-85-3, Isoniazide 55-22-1,
 Isonicotinic acid, biological studies 69-53-4, Ampicillin 443-48-1,
 Metronidazole 564-25-0, Doxycycline 6998-60-3, Rifamycin 8064-90-2,
 Bactrim 10118-90-8, Minocycline 13292-46-1, Rifampin 26787-78-0,
 Amoxicillin 36877-68-6, Nitroimidazole 81103-11-9, Clarithromycin
 82419-36-1, Ofloxacin 83905-01-5, Azithromycin
 (for Chlamydia infection treatment; diagnosis and management of
 infection caused by Chlamydia)

IT 52-67-5, Penicillamine
 (for Chlamydia infection treatment; diagnosis and management of
 infection caused by Chlamydia)

RN 52-67-5 USPATFULL

CN D-Valine, 3-mercaptop- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L221 ANSWER 23 OF 24 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
 ACCESSION NUMBER: 2004-449666 [42] WPIX
 DOC. NO. CPI: C2004-168507
 TITLE: Use of agent(s) that elevate intracellular cyclic
 adenosine monophosphate or intracellular calcium levels
 in neural tissue for modulating neurogenesis to treat
 central nervous system disorder.

DERWENT CLASS: B02 B03
 INVENTOR(S): BERTILSSON, G; ERLANDSSON, R; FRISEN, J; HAEGESTRAND, A;
 HAGGBLAD, J; HEIDRICH, J; HELLSTROM, K; JANSSON, K;
 KORTESMAA, J; LINDQUIST, P; LUNDH, H; MCGUIRE, J; MERCER,
 A; NJBERG, K; OSSOINAK, A; PATRONE, C; RONNHOLM, H;
 WIKSTROM, L; ZACHRISSON, O; HAEGBBLAD, J; HELLSTROEM, K;
 ROENNHOLM, H; WIKSTROEM, L; HAEGERSTRAND, A; HELLSTROM,
 N; NYBERG, K; HELLSTROEM, N
 (BERT-I) BERTILSSON G; (ERLA-I) ERLANDSSON R; (FRIS-I)
 FRISEN J; (HAEG-I) HAEGESTRAND A; (HAGG-I) HAGGBLAD J;
 (HEID-I) HEIDRICH J; (HELL-I) HELLSTROM K; (JANS-I)
 JANSSON K; (KORT-I) KORTESMAA J; (LIND-I) LINDQUIST P;
 (LUND-I) LUNDH H; (MCGU-I) MCGUIRE J; (MERC-I) MERCER A;
 (NEUR-N) NEURONOVA AB; (NJBE-I) NJBERG K; (OSSO-I)
 OSSOINAK A; (PATR-I) PATRONE C; (RONN-I) RONNHOLM H;
 (WIKS-I) WIKSTROM L; (ZACH-I) ZACHRISSON O; (HAEG-I)
 HAEGBBLAD J; (HELL-I) HELLSTROEM K; (ROEN-I) ROENNHOLM H;
 (WIKS-I) WIKSTROEM L

COUNTRY COUNT: 108
 PATENT INFORMATION:

PATENT NO	KIND DATE	WEEK	LA	PG	MAIN IPC
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11/06/2006

Handy 10/617943

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WO 2004045592 A2 20040603 (200442)* EN 77 A61K031-00
 RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE
 LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
 W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE
 DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
 KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM
 PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US
 UZ VC VN YU ZA ZM ZW
 AU 2003280117 A1 20040615 (200470) A61K031-00
 US 2005009742 A1 20050113 (200506) A61K038-17
 US 2005009847 A1 20050113 (200506) A61K038-17
 US 2005209142 A1 20050922 (200563) A61K038-22
 EP 1583541 A2 20051012 (200568) EN A61K031-675
 R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
 MC MK NL PT RO SE SI SK TR
 US 6969702 B2 20051129 (200578) A01N037-18
 US 2006079448 A1 20060413 (200626) A61K038-22
 JP 2006514630 W 20060511 (200635) 70 A61K045-00

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004045592	A2	WO 2003-IB5311	20031120
AU 2003280117	A1	AU 2003-280117	20031120
US 2005009742	A1 Provisional CIP of	US 2002-427912P US 2003-718071 US 2004-850055	20021120 20031120 20040519
US 2005009847	A1 Provisional	US 2002-427912P US 2003-718071	20021120 20031120
US 2005209142	A1 Provisional CIP of CIP of	US 2002-427912P US 2003-718071 US 2004-850055 US 2004-993667	20021120 20031120 20040519 20041119
EP 1583541	A2	EP 2003-772495 WO 2003-IB5311	20031120 20031120
US 6969702	B2 Provisional CIP of	US 2002-427912P US 2003-718071 US 2004-850055	20021120 20031120 20040519
US 2006079448	A1 Provisional CIP of Div ex	US 2002-427912P US 2003-718071 US 2004-850055 US 2005-288495	20021120 20031120 20040519 20051128
JP 2006514630	W	WO 2003-IB5311 JP 2004-553032	20031120 20031120

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003280117	A1 Based on	WO 2004045592
EP 1583541	A2 Based on	WO 2004045592
US 2006079448	A1 Div ex	US 6969702
JP 2006514630	W Based on	WO 2004045592

PRIORITY APPLN. INFO: US 2002-427912P 20021120; US
 2003-718071 20031120; US
 2004-850055 20040519; US
 2004-993667 20041119; US
 2005-288495 20051128

INT. PATENT CLASSIF.:

MAIN: A01N037-18; A61K031-00; A61K031-675; A61K038-17;
A61K038-22; A61K045-00

SECONDARY: A61K031-352; A61K031-4015; A61K031-522; A61K031-7042;
A61K031-7076; A61K035-66; A61K035-74; A61K038-00;
A61K038-12; A61K038-23; A61P009-00; A61P009-10;
A61P019-00; A61P025-00; A61P025-14; A61P025-16;
A61P025-28; A61P037-00; A61P043-00

BASIC ABSTRACT:

WO2004045592 A UPAB: 20040702

NOVELTY - In modulating neurogenesis in neural tissue of a patient exhibiting symptom(s) of central nervous system disorder, such as neurodegenerative, ischemic or learning and memory disorder or neurological trauma, at least one agent (A) that elevates intracellular cyclic adenosine monophosphate (cAMP) levels or at least one agent (B) that elevates intracellular Ca²⁺ levels in the neural tissue, is administered where (A) modulates and (B) induces neurogenesis.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) method (M1) for increasing the intracellular levels of cAMP in a cell or method (M2) for stimulating intracellular cAMP in a cell of a patient involving: contacting the cell, or administering to the patient an agent selected from (des-Arg9,Leu8)-bradykinin, (Des-Arg9)-bradykinin, alpha-neoendorphin, CART (61-102), DTLET, eledoisin, urotensin II, (NIe8, Tyr34)-parathyroid hormone (1-34) amide and/or (Cys3,6, Tyr8, Pro9)-Substance P; and

(2) modulating neurogenesis in vitro by:

(a) culturing a population of neural cells, comprising neural stem cells;
(b) adding at least one neurogenesis modulating agent to the cultured cells; and
(c) repeating step (a) and (b) to achieve a desired level of neurogenesis.

ACTIVITY - Nootropic; Neuroprotective; CNS-Gen.; Cerebroprotective; Vasotropic; Anticonvulsant; Antiparkinsonian; Hemostatic; Hypertensive; Muscular-Gen.; Ophthalmological; Antiinflammatory; Analgesic; Antidiabetic.

MECHANISM OF ACTION - Neurogenesis modulator; Neural stem or progenitor cell proliferation, differentiation and/or migration modulator; Neural tissue G-protein coupled receptor activator; Neurogenesis inducer; Intracellular neural cAMP enhancer; Intracellular neural cAMP stimulator; Intracellular neural Ca²⁺ enhancer.

N-6,2-O-Dibutyryl-adenosine (a) was studied for induction of proliferation and increase in the cAMP levels in mouse adult neural stem cells by adding to the culture at 100 nM. After 15 minutes cAMP levels were measured and after 4 days ATP levels were measured. Control cells were treated with vehicle. ATP Level (nM, ATP/well), fold induction of ATP, cAMP level (pmol/well) and fold induction of cAMP in the cells treated with (a)/vehicle were found to be: 13.9 plus or minus 1.1/9.3 plus or minus 0.6, 1.5/1, 0.1 plus or minus 0.01/0.02 plus or minus 0.01 and 4.5/-, respectively.

USE - For modulating neurogenesis in neural tissue of a patient exhibiting at least one symptom of central nervous system disorder, such as neurodegenerative disorder, ischemic disorder, neurological trauma and learning and memory disorder, e.g. Parkinson's disease and Parkinson's disorders, Huntington's disease, Alzheimer's disease, multiple sclerosis, amyotrophic lateral sclerosis, Shy-Drager syndrome, progressive supranuclear palsy, Lewy body disease, spinal ischemia, ischemic stroke, cerebral infarction, spinal cord injury, cancer-related brain and spinal cord injury, multi-infarct dementia and geriatric dementia; for increasing

the intracellular cAMP levels in a cell (preferably a cell from a neural tissue); for stimulating intracellular cAMP in a cell of a patient; and for in vitro modulation of neurogenesis (claimed). Also useful for the treatment of idiopathic orthostatic hypotension, structural lesions of cerebellum (e.g. those associated with infarcts, hemorrhage or tumors), spinocerebellar degenerations (e.g. associated with Friedreich's ataxia), abetalipoproteinemia, Refsum's disease, cerebellar ataxia, multiple systems atrophy, **ataxia-telangiectasia**, mitochondrial multisystem disorders, acute disseminated encephalomyelitis, adrenoleukodystrophy, adrenomyeloneuropathy, Leber's hereditary optic dystrophy, HTLV-associated myelopathy, progressive bulbar palsy, progressive muscular atrophy, primary lateral sclerosis, progressive pseudobulbar palsy, spinal muscular atrophy, plexopathy, acute branchial neuritis, peripheral neuropathy, multiple mononeuropathies, polyneuropathies, nerve palsy, carpal tunnel syndrome, peroneal nerve palsy, radial nerve palsy, Guillain-Barre syndrome, chronic relapsing, hereditary motor and sensory neuropathy, myasthenia gravis, neuro-ophthalmological disorders, cranial nerve palsies, trigeminal neuralgia, Bell's palsy, glossopharyngeal neuralgia, radiation-induced injury of the nervous system, chemotherapy-induced neuropathy, taxol neuropathy, vincristine neuropathy, diabetic neuropathy, autonomic neuropathy, polyneuropathy, mononeuropathy, transient ischemic attacks, subclavian steal syndrome, drop attacks, ischemic stroke, hemorrhagic stroke and brain infarction; for the detection of endogenous agents in cells that are involved in the mediation of signal transduction pathways in the regulation of neurogenesis function; and in the diagnosis of diseases and determine the role of stem and progenitor cells in the disease.

ADVANTAGE - The agent modulates neurogenesis in neural tissue by modulating proliferation, differentiation, migration or survival of neural stem cells or progenitor cells in the tissue; by maintaining or increasing the amount or percentage of doublecortin positive cells in the neural tissue relative to a patient not dosed with the agent or by activation of a G-protein coupled receptor in the neural tissue. The method results in elevation of cAMP levels of the neural stem cells by at least 20% compared to untreated tissue. The *in vivo* induction of neurogenesis allows treatment of disorders caused by cell loss, injury or disease by endogenous replacement and obviates the need for transplanting foreign cells into a patient. Neurogenesis can also be induced by administration of the neurogenesis-modulating agent directly into a desired site, which avoids unnecessary systemic administration and possible side effects and further provides an alternative to the use of drugs and the controversial use of large quantities of embryonic tissue for treatment of Parkinson's disease.

Dwg.0/1

FILE SEGMENT: CPI
FIELD AVAILABILITY: AB; DCN
MANUAL CODES: CPI: B04-A01; B04-A03; B04-A04; B04-A05; B04-A06;
B04-B03B; B04-C01; B04-H03; B04-J01; B04-J03B;
B04-J04A; B04-J04B; B04-J05; B04-J07; B04-J08;
B04-J09; B04-J11; B04-J13; B05-B01M; B06-H; B07-H;
B10-B02E; B10-B02G; B10-B03B; B10-B04B; B10-C04B;
B10-E02; B12-K04; B14-F02B; B14-F02D; B14-J01A3;
B14-J01A4; B14-J02; B14-J05; B14-L01; B14-N03;
B14-N16; B14-S01
AN 2004-449666 [42] WPIX
DC B02 B03
IC ICM A01N037-18; A61K031-00; A61K031-675; A61K038-17; A61K038-22;
A61K045-00
ICS A61K031-352; A61K031-4015; A61K031-522; A61K031-7042; A61K031-7076;

A61K035-66; A61K035-74; A61K038-00; A61K038-12; A61K038-23;
 A61P009-00; A61P009-10; A61P019-00; A61P025-00; A61P025-14;
 A61P025-16; A61P025-28; A61P037-00; A61P043-00

MC CPI: B04-A01; B04-A03; B04-A04; B04-A05; B04-A06; B04-B03B; B04-C01;
 B04-H03; B04-J01; B04-J03B; B04-J04A; B04-J04B; B04-J05; B04-J07;
 B04-J08; B04-J09; B04-J11; B04-J13; B05-B01M; B06-H; B07-H; B10-B02E;
 B10-B02G; B10-B03B; B10-B04B; B10-C04B; B10-E02; B12-K04; B14-F02B;
 B14-F02D; B14-J01A3; B14-J01A4; B14-J02; B14-J05; B14-L01; B14-N03;
 B14-N16; B14-S01

DRN 0018-U; 0026-U; 0066-U; 0107-U; 0152-U; 0163-U; 0171-U; 0192-U; 0501-U;
 0971-U; 1274-U; 1361-U; 1393-U; 1449-U; 1553-U; 1874-U; 1876-U; 1969-U;
 2007-U; 2026-U; 2032-U

CMC UPB 20040702

M1 *01* D011 D601 F012 F014 F423 F521 G010 G013 G100 H1 H100 H101 H181
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 J111 J171 J172 J3 J311 J371 K0 K2 K224 L2 L250 M210 M211
 M271 M280 M281 M311 M312 M313 M314 M315 M320 M321 M322 M331 M332
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 M521 M530 M531 M540 M620 M781 M904 M905 M910 P444 P446 P510 P517
 P525 P526 P528 P617 P922
 DCN: R01874-K; R01874-T; R01874-U

M1 *02* M423 M781 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA00F4-K; RA00F4-T; RA00F4-U

M1 *03* M423 M781 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA0GBQ-K; RA0GBQ-T; RA0GBQ-U

M1 *04* M423 M781 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA05BP-K; RA05BP-T; RA05BP-U

M1 *05* M423 M781 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RAAEIC-K; RAAEIC-T; RAAEIC-U

M1 *06* D011 D601 F014 F016 F750 G010 G013 G100 H1 H101 H182 H4 H402
 H441 H481 H8 J0 J014 J1 J172 J3 J311 J321 J373 J5 J523
 L9 L941 L999 M1 M123 M126 M129 M132 M139 M280 M311 M312 M313
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 RIN: 00598
 DCN: R10548-K; R10548-T; R10548-U

M1 *07* F011 F012 F014 F016 F017 F423 F750 G013 G100 H1 H100 H121 H2
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 M522 M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
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 RIN: 00598
 DCN: R06740-K; R06740-T; R06740-U

M1 *08* F011 F012 F014 F016 F423 F750 G010 G013 G100 H1 H101 H121 H181
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M1 *09* F014 F521 G010 G100 H1 H100 H181 H4 H401 H481 H8 J0 J011
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 DCN: R01553-K; R01553-T; R01553-U

M1 *10* F012 F423 G010 G013 G100 H1 H100 H101 H181 H182 H4 H401 H441
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M1 *11* D011 D601 F011 F012 F014 F019 F423 F499 F521 G010 G019 G100 H1
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M1 *14* D011 D601 F014 F521 G010 G013 G100 H1 H100 H101 H181 H182 H4
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 DCN: R01876-K; R01876-T; R01876-U

M1 *15* D011 D601 F014 F521 G010 G013 G019 G100 H1 H101 H183 H4 H405
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M1 *16* F012 F423 G010 G013 G100 H1 H100 H101 H181 H182 H4 H401 H441
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M1 *17* D011 D601 F011 F012 F014 F019 F423 F499 F521 G010 G013 G019 G100
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 DCN: R01836-K; R01836-T; R01836-U

M1 *18* D011 D601 G010 G013 G100 H1 H100 H181 H5 H598 H9 J0 J011
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 DCN: R10850-K; R10850-T; R10850-U

M1 *19* F011 F012 F019 F423 F499 G010 G100 H1 H100 H181 H2 H213 H4
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M1 *20* D011 D601 F014 F521 G010 G013 G100 H1 H101 H182 H4 H404 H441
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M1 *21* F011 F012 F014 F019 F423 F499 F521 F599 G010 G100 H1 H101 H183
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M1 *23* F011 F012 F015 F019 F423 F499 G010 G100 H1 H100 H181 H2 H211
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M1 *24* F014 F521 G010 G100 H1 H101 H182 H4 H402 H482 H5 H598 H8
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M2 *25* D015 D932 H2 H212 J5 J522 L9 L910 M210 M211 M273 M282 M320
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 P525 P528 P617 P922
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M2 *26* D011 D015 D932 H1 H181 H2 H201 H212 J5 J522 L9 L910 M210
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M2 *27* D014 D023 D120 G015 G100 H4 H404 H405 H421 H444 H8 J5 J521
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 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R00971-K; R00971-T; R00971-U;
RA0055-K; RA0055-T; RA0055-U

M2 *28* D011 D015 D932 H1 H181 H2 H201 H212 J5 J522 J581 L9 L910
 M210 M211 M213 M231 M262 M273 M281 M282 M314 M321 M332 M342 M381
 M391 M412 M511 M520 M530 M540 M781 M904 M905 P444 P446 P510 P517
 P525 P528 P617 P922
 DCN: R20131-K; R20131-T; R20131-U

M2 *29* D015 D932 H2 H212 J5 J522 L9 L910 M210 M211 M214 M232 M273
 M282 M320 M412 M511 M520 M530 M540 M781 M904 M905 P444 P446 P510
 P517 P525 P526 P528 P617 P922
 DCN: R21071-K; R21071-T; R21071-U

M2 *30* F012 F014 F015 F522 G013 G100 H5 H594 H9 J5 J521 J581 L9
 L921 M1 M123 M131 M210 M211 M240 M271 M281 M320 M413 M510 M521
 M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617
 P922
 DCN: R11079-K; R11079-T; R11079-U

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M2 *31* D023 D210 M210 M211 M214 M232 M240 M282 M320 M412 M511 M520 M530
M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: RAAWOS-K; RAAWOS-T; RAAWOS-U

M2 *32* D011 D023 D631 G015 G100 H5 H543 H8 M1 M123 M132 M210 M211
M272 M283 M311 M321 M342 M412 M511 M520 M531 M540 M640 M781 M904
M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R04935-K; R04935-T; R04935-U

M2 *33* C017 C100 C720 C800 C801 C803 C804 C805 C806 C807 F011 F013 F522
G013 G015 G019 G100 H1 H181 H2 H201 H5 H581 H6 H602 H609
H643 H8 K0 L7 L721 M1 M121 M129 M132 M139 M150 M280 M311
M312 M321 M322 M332 M342 M343 M373 M393 M411 M510 M521 M533 M540
M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: RA7PW6-K; RA7PW6-T; RA7PW6-U

M2 *34* F013 F014 F016 F530 G013 G100 J5 J521 K0 L1 L110 L2 L250
L9 L941 M1 M113 M210 M211 M240 M273 M281 M320 M413 M510 M521
M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: RA06CF-K; RA06CF-T; RA06CF-U

M2 *35* D015 D931 H1 H101 H182 H2 H212 J5 J522 L9 L910 M210 M211
M273 M282 M312 M321 M332 M342 M383 M391 M412 M511 M520 M530 M540
M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R03933-K; R03933-T; R03933-U

M2 *36* G015 G019 G100 H4 H404 H444 H7 H721 H8 J5 J581 M1 M121
M135 M280 M312 M321 M332 M342 M372 M391 M414 M510 M520 M532 M540
M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R19932-K; R19932-T; R19932-U

M2 *37* C017 C100 C800 C801 C803 C804 C805 C806 C807 D011 D013 D830 H2
H211 J0 J011 J2 J211 K0 K6 K630 M210 M211 M212 M263 M272
M273 M281 M282 M320 M411 M511 M520 M530 M540 M640 M781 M904 M905
P444 P446 P510 P517 P525 P526 P528 P617 P922
RIN: 01190
DCN: R11856-K; R11856-T; R11856-U

M2 *38* C017 C100 C800 C801 C803 C804 C805 C806 C807 D011 D022 E800 F011
F014 F553 H1 H183 H2 H203 H6 H685 M210 M211 M273 M281 M311
M313 M321 M332 M342 M344 M353 M383 M391 M411 M511 M521 M530 M540
M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: RA6KYT-K; RA6KYT-T; RA6KYT-U

M2 *39* F012 F013 F014 F015 F016 F431 F432 J5 J521 K0 L1 L142 L9
L941 M1 M116 M210 M211 M240 M281 M320 M413 M510 M522 M530 M540
M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R11076-K; R11076-T; R11076-U

M2 *40* D013 D016 D023 D026 D029 D030 D220 H4 H403 H421 H462 H7 H715
H721 H8 J0 J011 J2 J261 J5 J521 M210 M211 M212 M240 M262
M281 M283 M320 M412 M511 M520 M530 M540 M781 M800 M904 M905 P444
P446 P510 P517 P525 P526 P528 P617 P922
RIN: 03577
DCN: R04356-K; R04356-T; R04356-U

M2 *41* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D030
D160 D931 H1 H100 H122 H2 H201 J0 J012 J2 J222 L910 M210
M213 M231 M262 M282 M320 M411 M512 M520 M530 M540 M781 M904 M905
P444 P446 P510 P517 P525 P526 P528 P617 P922
RIN: 08000
DCN: RADLVX-K; RADLVX-T; RADLVX-U

M2 *42* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D160
D931 H1 H100 H122 H2 H201 H4 H401 H421 H6 H602 H621 H8
L943 M280 M320 M411 M512 M520 M530 M540 M781 M904 M905 P444 P446
P510 P517 P525 P526 P528 P617 P922
RIN: 08000 08000
DCN: R18084-K; R18084-T; R18084-U; R18085-K; R18085-T; R18085-U

M2 *43* B615 B701 B713 B720 B815 B831 B840 D011 D012 D013 D016 D019 D160

D931 H1 H100 H122 H2 H201 H4 H401 H421 H6 H603 H621 H8
 L943 M280 M320 M411 M512 M520 M530 M540 M781 M904 M905 P444 P446
 P510 P517 P525 P526 P528 P617 P922
 RIN: 08000
 DCN: RA04NE-K; RA04NE-T; RA04NE-U

M2 *44* D011 D024 D640 G013 G100 H403 H443 H602 H641 K431 K432 M113 M210
 M211 M271 M280 M281 M320 M412 M510 M511 M520 M530 M531 M540 M620
 M650 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 RIN: 01843
 DCN: RAEJ8B-K; RAEJ8B-T; RAEJ8B-U

M2 *45* C017 C100 C800 C801 C803 C804 C805 C806 C807 G015 G100 H1 H100
 H181 H4 H402 H442 H8 M280 M312 M321 M332 M342 M373 M391 M411
 M510 M520 M531 M540 M640 M781 M904 M905 P444 P446 P510 P517 P525
 P526 P528 P617 P922
 DCN: R11187-K; R11187-T; R11187-U

M2 *46* C017 C100 C720 C800 C801 C803 C804 C805 C806 C807 D011 D023 D030
 E310 H1 H181 H2 H201 H4 H402 H442 H8 M210 M211 M273 M280
 M281 M320 M411 M511 M520 M530 M540 M640 M781 M904 M905 P444 P446 P510 P517
 P525 P526 P528 P617 P922
 RIN: 05171
 DCN: R22680-K; R22680-T; R22680-U

M2 *47* B215 B713 B720 B819 B831 C108 C720 C800 C801 C802 C803 C804 C805
 C807 F014 F521 H1 H100 H181 M280 M312 M321 M332 M342 M373 M391
 M411 M510 M521 M530 M540 M640 M781 M904 M905 P444 P446 P510 P517
 P525 P526 P528 P617 P922
 DCN: R12275-K; R12275-T; R12275-U

M2 *48* C316 D011 D022 D601 H103 H181 J012 J172 K353 M210 M211 M273 M280
 M283 M311 M312 M321 M332 M342 M373 M382 M391 M392 M412 M511 M520
 M530 M540 M620 M650 M781 M904 M905 P444 P446 P510 P517 P525 P526
 P528 P617 P922
 DCN: RA4D8G-K; RA4D8G-T; RA4D8G-U

M2 *49* G037 G553 H4 H403 H462 H481 H7 H722 H8 J0 J011 J1 J171
 M280 M315 M322 M331 M332 M342 M372 M373 M391 M415 M510 M520 M530
 M541 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617
 P922
 DCN: R01361-K; R01361-T; R01361-U

M2 *50* G037 G553 H4 H402 H461 H481 H7 H721 H8 J0 J011 J1 J171
 J5 J561 M280 M315 M322 M331 M332 M342 M372 M373 M391 M415 M510
 M520 M530 M541 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526
 P528 P617 P922
 DCN: R01449-K; R01449-T; R01449-U; R10058-K; R10058-T; R10058-U

M2 *51* D011 D023 D130 H4 H402 H461 H481 H8 J0 J011 J1 J171 M280
 M314 M315 M321 M331 M332 M342 M372 M373 M391 M412 M511 M520 M530
 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 RIN: 01010
 DCN: R03935-K; R03935-T; R03935-U

M2 *52* G032 G033 G060 G630 H100 H181 H4 H402 H403 H461 H481 H483 H720
 H721 H731 H8 J011 J171 M280 M314 M316 M321 M332 M333 M334 M342
 M343 M344 M372 M373 M383 M391 M415 M510 M520 M530 M541 M620 M650
 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RAEJ8C-K; RAEJ8C-T; RAEJ8C-U

M2 *53* G037 G553 H4 H402 H461 H481 H7 H721 H8 J0 J011 J2 J271
 J5 J561 M210 M211 M272 M281 M315 M316 M321 M332 M333 M342 M372
 M373 M391 M415 M510 M520 M530 M541 M781 M904 M905 P444 P446 P510
 P517 P525 P526 P617 P922
 DCN: R21800-K; R21800-T; R21800-U

M2 *54* A111 A960 B615 B702 B713 B720 B795 B815 B833 C710 D011 D019 D931
 F012 F013 F014 F015 F113 H1 H100 H122 H2 H201 H4 H402 H422
 H8 L943 M280 M311 M321 M342 M373 M391 M411 M511 M521 M530 M540
 M630 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922

DCN: R18980-K; R18980-T; R18980-U
M2 *55* D021 D601 H1 H102 H181 H4 H401 H481 H5 H541 H8 M210 M213
M232 M273 M281 M313 M321 M332 M343 M383 M391 M412 M511 M520 M530
M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617
P922
DCN: R01969-K; R01969-T; R01969-U
M2 *56* F011 F013 F014 F433 G013 G017 G100 H1 H100 H141 H181 H2 H201
H5 H521 H542 H6 H601 H602 H642 H8 J0 J011 J3 J321 M1
M123 M136 M210 M211 M272 M282 M313 M321 M332 M342 M383 M391 M413
M510 M521 M532 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526
P528 P617 P922
DCN: RA0GIN-K; RA0GIN-T; RA0GIN-U
M2 *57* F012 F522 G012 G013 G100 H103 H141 H401 H441 K431 K432 M121 M143
M210 M211 M240 M271 M281 M311 M320 M321 M342 M373 M391 M413 M510
M520 M521 M530 M532 M540 M620 M650 M781 M904 M905 P444 P446 P510
P517 P525 P526 P528 P617 P922
DCN: RA0M8Z-K; RA0M8Z-T; RA0M8Z-U
M2 *58* F011 F012 F013 F423 G010 G017 G100 H1 H102 H141 H181 H2 H201
H5 H541 H6 H602 H641 H8 J0 J011 J3 J321 M1 M123 M136
M210 M211 M240 M272 M273 M281 M311 M321 M342 M373 M391 M413 M510
M521 M532 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
P617 P922
DCN: RA61W6-K; RA61W6-T; RA61W6-U
M2 *59* D011 D022 E240 F011 F014 F553 H1 H121 H181 H2 H202 H6 H602
H641 L943 M210 M211 M273 M281 M320 M412 M511 M521 M530 M540 M781
M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
RIN: 03676
DCN: R22668-K; R22668-T; R22668-U
M2 *60* D013 D022 D601 F011 F012 F014 F019 F433 F523 G013 G100 H1 H141
H181 H2 H202 H211 H6 H601 H602 H642 J5 J521 L9 L921 M1
M116 M280 M312 M321 M332 M342 M383 M391 M412 M511 M522 M531 M540
M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R20437-K; R20437-T; R20437-U
M2 *61* D011 D012 E850 F011 F014 F553 H1 H121 H181 H2 H202 L943 M210
M211 M240 M273 M281 M320 M412 M511 M521 M530 M540 M781 M904 M905
P444 P446 P510 P517 P525 P526 P528 P617 P922
RIN: 46639
DCN: RA04JZ-K; RA04JZ-T; RA04JZ-U
M2 *62* D011 D014 D022 D790 E410 F011 F014 F433 H1 H181 H2 H201 H6
H601 H641 J5 J521 L9 L941 M1 M116 M210 M211 M240 M281 M312
M321 M332 M342 M373 M391 M412 M512 M521 M530 M540 M781 M904 M905
P444 P446 P510 P517 P525 P526 P528 P617 P922
RIN: 01123 01608
DCN: RA07U9-K; RA07U9-T; RA07U9-U
M2 *63* C316 F011 F012 F423 G015 G100 H1 H181 H2 H201 H5 H541 H8
J0 J011 J3 J331 K0 K3 K353 M210 M211 M212 M272 M273 M281
M311 M321 M342 M373 M391 M413 M510 M521 M531 M540 M781 M904 M905
P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R06022-K; R06022-T; R06022-U; R16364-K; R16364-T; R16364-U
M2 *64* D011 D022 E800 H1 H103 H182 H2 H201 H6 H602 H641 M210 M211
M273 M282 M313 M321 M332 M342 M383 M391 M412 M511 M520 M530 M540
M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R00026-K; R00026-T; R00026-U; R08400-K; R08400-T; R08400-U
M2 *65* F011 F014 F017 F433 G013 G019 G100 H1 H181 H2 H201 H4 H401
H421 H6 H601 H602 H642 H8 J5 J581 M1 M113 M280 M313 M321
M332 M342 M381 M391 M413 M510 M521 M532 M540 M781 M904 M905 M910
P444 P446 P510 P517 P525 P526 P528 P617 P922
DCN: R00066-K; R00066-T; R00066-U
M2 *66* D013 D019 D022 D712 D799 F011 F014 F433 H1 H181 H2 H201 H212
H6 H602 H641 J5 J522 L9 L921 L999 M280 M313 M321 M332 M342

M383 M391 M412 M512 M521 M530 M540 M781 M904 M905 P444 P446 P510
 P517 P525 P526 P528 P617 P922
 DCN: R06637-K; R06637-T; R06637-U; R14531-K; R14531-T; R14531-U

M2 *67* D011 D022 E800 F011 F014 F553 H1 H183 H2 H203 H6 H685 J0
 J011 J2 J271 M220 M223 M231 M262 M281 M312 M313 M321 M332 M342
 M383 M392 M412 M511 M521 M530 M540 M781 M904 M905 P444 P446 P510
 P517 P525 P526 P528 P617 P922
 DCN: R04431-K; R04431-T; R04431-U

M2 *68* D014 D740 F011 F014 F433 G013 G100 H1 H181 H2 H201 H211 H6
 H601 H641 J5 J522 J581 L9 L910 M1 M123 M131 M280 M312 M321
 M332 M342 M383 M391 M412 M511 M521 M531 M540 M781 M904 M905 P444
 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R12049-K; R12049-T; R12049-U

M2 *69* D011 D013 E320 G010 G100 H1 H182 H2 H202 K0 L4 L463 M210
 M211 M273 M282 M311 M322 M342 M373 M392 M412 M511 M520 M531 M540
 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R17747-K; R17747-T; R17747-U

M2 *70* C017 C100 C800 C801 C803 C804 C805 C806 C807 D013 D023 D740 F011
 F012 F014 F111 F553 H1 H100 H122 H2 H201 H211 H5 H542 H8
 J0 J011 J3 J311 L910 M210 M211 M272 M282 M320 M411 M511 M522
 M530 M540 M640 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
 P617 P922
 DCN: R07015-K; R07015-T; R07015-U

M2 *71* D023 E340 H4 H401 H461 H8 J0 J011 J2 J251 M210 M211 M272
 M281 M320 M412 M511 M520 M530 M540 M781 M904 M905 P444 P446 P510
 P517 P525 P526 P528 P617 P922
 DCN: R17247-K; R17247-T; R17247-U

M2 *72* D011 D014 D932 H1 H181 H2 H201 H211 J5 J522 L9 L910 M210
 M211 M273 M282 M320 M412 M511 M520 M530 M540 M781 M904 M905 M910
 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R00501-K; R00501-T; R00501-U

M2 *73* F012 F013 F014 F015 F431 F432 H1 H100 H121 J5 J521 L9 L941
 M1 M116 M280 M320 M413 M510 M522 M530 M540 M781 M904 M905 P444
 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R11075-K; R11075-T; R11075-U

M2 *74* D011 D021 D029 D030 E550 G030 G530 H1 H181 H2 H201 H4 H402
 H421 H441 H8 J5 J561 M280 M311 M321 M342 M373 M391 M412 M511
 M520 M530 M541 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528
 P617 P922
 DCN: R07064-K; R07064-T; R07064-U; R15129-K; R15129-T; R15129-U

M2 *75* D011 D021 D029 D030 E550 H1 H181 H2 H201 H4 H402 H421 H441
 H7 H716 H721 H8 J5 J561 M210 M213 M231 M273 M281 M320 M412
 M511 M520 M530 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525
 P526 P528 P617 P922
 DCN: R01274-K; R01274-T; R01274-U; R15119-K; R15119-T; R15119-U

M2 *76* G015 G100 H1 H102 H181 H4 H403 H441 H482 H8 M210 M214 M233
 M273 M281 M311 M312 M321 M332 M342 M343 M373 M392 M414 M510 M520
 M531 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528
 P617 P922
 DCN: R02007-K; R02007-T; R02007-U; R06679-K; R06679-T; R06679-U

M2 *77* G015 G100 H1 H102 H181 H4 H403 H441 H482 H8 M210 M214 M233
 M273 M281 M311 M312 M321 M332 M342 M343 M373 M392 M414 M510 M520
 M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617
 P922
 DCN: RA1EEJ-K; RA1EEJ-T; RA1EEJ-U

M2 *78* G016 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M213 M232
 M273 M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540
 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R01393-K; R01393-T; R01393-U; R06678-K; R06678-T; R06678-U

M2 *79* G016 G100 H1 H102 H181 H4 H403 H442 H481 M210 M214 M233 M273

M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540 M781
 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R02026-K; R02026-T; R02026-U; R14963-K; R14963-T; R14963-U

M2 *80* F012 F013 F016 F432 H1 H102 H181 H4 H402 H482 H8 J5 J521
 M210 M214 M233 M273 M281 M311 M312 M321 M332 M342 M343 M373 M392
 M413 M510 M521 M530 M540 M781 M904 M905 P444 P446 P510 P517 P525
 P526 P528 P617 P922
 DCN: R06393-K; R06393-T; R06393-U; R12431-K; R12431-T; R12431-U

M2 *81* G010 G015 G100 H1 H102 H181 H4 H403 H441 H482 H5 H581 H8
 M280 M311 M312 M314 M315 M321 M332 M342 M343 M373 M383 M391 M393
 M414 M510 M520 M532 M540 M781 M904 M905 P444 P446 P510 P517 P525
 P526 P528 P617 P922
 DCN: R16589-K; R16589-T; R16589-U; R18850-K; R18850-T; R18850-U

M2 *82* G013 G015 G019 G100 H1 H102 H181 H4 H401 H481 H8 J0 J012
 J2 J232 M1 M121 M129 M136 M139 M210 M211 M214 M233 M240 M273
 M281 M282 M312 M321 M332 M343 M373 M391 M414 M510 M520 M533 M540
 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R04238-K; R04238-T; R04238-U

M2 *83* G015 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M214 M233
 M273 M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540
 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R04844-K; R04844-T; R04844-U

M2 *84* G013 G015 G100 H1 H102 H181 H4 H403 H443 H8 M280 M312 M314
 M321 M331 M332 M342 M373 M392 M414 M510 M520 M532 M540 M781 M904
 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R11507-K; R11507-T; R11507-U; R13752-K; R13752-T; R13752-U

M2 *85* G015 G100 H1 H100 H181 H4 H402 H442 H8 J0 J011 J1 J171
 M280 M313 M321 M331 M343 M349 M371 M391 M414 M510 M520 M531 M540
 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA0107-K; RA0107-T; RA0107-U

M2 *86* G015 G100 H1 H100 H181 H4 H402 H442 H8 J0 J011 J1 J171
 M280 M312 M321 M332 M343 M349 M371 M391 M414 M510 M520 M531 M540
 M781 M800 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528 P617
 P922
 DCN: R12227-K; R12227-T; R12227-U

M2 *87* D011 D013 D019 D021 D030 E350 H1 H181 H2 H201 H4 H402 H421
 H461 H8 M210 M211 M212 M240 M273 M281 M320 M412 M511 M520 M530
 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 RIN: 09454
 DCN: R03739-K; R03739-T; R03739-U

M2 *88* D011 D019 D022 D621 D680 H4 H401 H481 H5 H541 H7 H715 H721
 H8 M1 M126 M132 M210 M211 M212 M240 M272 M281 M311 M321 M343
 M373 M391 M412 M512 M520 M530 M540 M781 M904 M905 M910 P444 P446
 P510 P517 P525 P526 P528 P617 P922
 DCN: R00107-K; R00107-T; R00107-U

M2 *89* D011 D860 F011 F014 F553 G011 G100 H1 H141 H181 H2 H202 M210
 M211 M240 M281 M312 M321 M332 M342 M373 M391 M412 M511 M521 M531
 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 RIN: 01094 01094
 DCN: R14842-K; R14842-T; R14842-U; R14843-K; R14843-T; R14843-U

M2 *90* G015 G100 H5 H581 H6 H602 H608 H642 H8 J0 J013 J1 J171
 J3 J331 J371 M210 M211 M215 M231 M272 M273 M281 M313 M322 M332
 M342 M343 M349 M381 M383 M391 M414 M510 M520 M531 M540 M781 M904
 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RAEAB8-K; RAEAB8-T; RAEAB8-U

M2 *91* F011 F012 F423 H2 H211 H4 H498 H9 J0 J012 J1 J111 J3
 J371 M280 M313 M321 M331 M342 M381 M391 M413 M510 M521 M530 M540
 M781 M800 M904 M905 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R04150-K; R04150-T; R04150-U; R07095-K; R07095-T; R07095-U

M2 *92* G010 G037 G111 G553 H4 H403 H462 H481 H7 H721 H8 J0 J011

J2 J271 M1 M123 M135 M210 M213 M232 M272 M281 M315 M322 M332
 M342 M343 M372 M373 M391 M414 M510 M520 M531 M541 M781 M904 M905
 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA01PF-K; RA01PF-T; RA01PF-U

M2 *93* G010 G021 G111 G221 H1 H100 H181 H7 H721 J0 J013 J3 J373
 M210 M211 M273 M283 M312 M315 M321 M322 M332 M333 M342 M343 M349
 M371 M381 M391 M392 M414 M510 M520 M532 M540 M781 M904 M905 P444
 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: RA1459-K; RA1459-T; RA1459-U

M2 *94* F011 F012 F014 F015 F019 F423 F499 F521 H2 H211 J0 J013 J3
 J312 J371 J5 J521 L9 L941 M280 M312 M321 M332 M343 M349 M371
 M391 M413 M510 M523 M530 M540 M781 M904 M905 M910 P444 P446 P510
 P517 P525 P526 P528 P617 P922
 DCN: R02032-K; R02032-T; R02032-U

M2 *95* D011 D014 D670 G010 G100 H1 H181 H2 H201 J0 J012 J2 J211
 J221 M1 M123 M136 M210 M211 M272 M273 M281 M320 M412 M511 M520
 M531 M540 M781 M904 M905 M910 P444 P446 P510 P517 P525 P526 P528
 P617 P922
 DCN: R00018-K; R00018-T; R00018-U; R11462-K; R11462-T; R11462-U

M2 *96* G010 G100 H1 H103 H181 J5 J581 M210 M212 M273 M282 M312 M321
 M331 M340 M342 M349 M381 M391 M414 M510 M531 M540 M781 M904 M905
 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R17080-K; R17080-T; R17080-U

M2 *97* F011 F012 F013 F014 F433 F541 H1 H101 H123 H2 H201 K0 K7
 K742 L9 L910 M280 M320 M413 M510 M522 M530 M540 M781 M904 M905
 P444 P446 P510 P517 P525 P526 P528 P617 P922
 DCN: R04592-K; R04592-T; R04592-U; R10186-K; R10186-T; R10186-U

M2 *98* C316 F012 F431 G013 G015 G100 H4 H401 H441 H8 J0 J011 J1
 J131 K0 K3 K353 K5 K534 L943 M1 M121 M123 M145 M147 M280
 M320 M413 M510 M521 M532 M540 M781 M904 M905 P444 P446 P510 P517
 P525 P526 P528 P617 P922
 DCN: R12996-K; R12996-T; R12996-U

M2 *99* G010 G100 H1 H100 H181 M280 M313 M321 M331 M342 M373 M391 M414
 M510 M520 M531 M540 M781 M904 M905 P444 P446 P510 P517 P525 P526
 P528 P617 P922
 DCN: RA04XY-K; RA04XY-T; RA04XY-U

L221 ANSWER 24 OF 24 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2003-341309 [32] WPIX

DOC. NO. CPI: C2003-089517

TITLE: New orthomolecular vitamin E derivatives useful for the treatment of e.g. cancer.

DERWENT CLASS: B02 D21 E13

INVENTOR(S): WILBURN, M D

PATENT ASSIGNEE(S): (WILB-I) WILBURN M D

COUNTRY COUNT: 1

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN	IPC
US 2003007961	A1	20030109	(200332)*		28	A61K038-44	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2003007961	A1	US 2001-886472	20010622

PRIORITY APPLN. INFO: US 2001-886472 20010622

INT. PATENT CLASSIF.:

MAIN: A61K038-44

SECONDARY: A61K031-714; A61K031-726

BASIC ABSTRACT:

US2003007961 A UPAB: 20030522

NOVELTY - Orthomolecular vitamin E derivatives (I) are new.

DETAILED DESCRIPTION - Orthomolecular vitamin E derivatives of formula (I), their salts, esters and solvates are new.

dotted line = optional double bond;

A, B, D, E = H or methyl;

R = reaction product derived from Q1, Q2 or phenyl (optionally substituted by 1-5 Q3);

Q1 = e.g. (flava-3-ol)n, alpha -ketoglutaric acid, alanine, flavin coenzymes (such as flavin mononucleotide or flavin adenine dinucleotide), para-amino benzoic acid (PABA) or zeaxanthin;

n = 1-12;

Q2 = 1-30C alkyl, 2-30C alkenyl or 2-30C alkynyl (all optionally substituted by 1-12 OH, carboxy, amino, halo, nitro, sulfhydryl or J);

J = phenyl or 5-7 membered heterocyclic ring (containing at least one O, N or S) (both optionally substituted by 1-5 OH, carboxy, halo, nitro, amino, sulfhydryl, methyl, 2-10C alkyl, 2-10C alkenyl, 2-10C alkynyl, methoxy, 2-8C alkoxy or -OC(O)R2 (all optionally substituted, and in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S));

R2 = trifluoromethyl, methyl, 1-10C alkyl, 2-10C alkenyl or 2-10C alkynyl (in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S);

Q3 = OH, carboxy, amino, halo, nitro, sulfhydryl, trifluoromethyl, methyl, 1-8C alkyl, 2-8C alkenyl, 2-8C alkynyl or 2-8C alkoxy (all optionally substituted, and in which at least one C of alkyl, alkenyl or alkynyl is optionally replaced by N, O or S).

The stereochemistry at each of the 2', 4' and 8' positions is R or S.

Full definitions are given in the DEFINITIONS (Full Definitions)

field.

ACTIVITY - Analgesic; Ophthalmological; Antiinflammatory; Cytostatic; Anorectic; Tranquilizer; Antidepressant; Nootropic; Neuroprotective; Antiparkinsonian; Hepatotropic; Antialcoholic; Cardiant; Antiarthritic; Osteopathic; Antirheumatic; Immunosuppressive; Dermatological; Vasotropic; Antithyroid; Antipsoriatic; Nephrotropic; Antidiabetic; Cerebroprotective; Anti-HIV; Antiarteriosclerotic; Gastrointestinal; Relaxant; Vasotropic; Antisickling; Respiratory; Anticoagulant; Gynecological; Hemostatic; Antiasthmatic; Antigout; Antianemic.

MECHANISM OF ACTION - Platelet Aggregation Inhibitor; Hydroxymethylglutaryl Coenzyme-A (HMG CoA) Reductase Inhibitor.

USE - For effecting a biological activity in an animal, such as aging, longevity, nerve activity, hematopoiesis, maintenance of blood cells, hepatic activity, nephritic activity, heart and cardiovascular function, pulmonary function, muscular function, cartilage health, bone health, joint health, gastrointestinal function, reproductive system function, vision, immune function, cell membrane integrity, pain and inflammation; for treating and preventing cancer, obesity, anxiety, depression, depression secondary to a chronic disease, Alzheimer's disease, Parkinson's disease, demyelinating disorder, peripheral neuropathy, enhancing mood and behavior, cirrhosis, chronic liver disease, alcoholic liver damage, toxic chemical exposure, NSAID-liver damage, estrogen induced liver problems, bile disorder, environmental chemical hypersensitivity, heart and/or artery disease risk due to elevated blood levels of homocysteine, osteoarthritis, rheumatoid arthritis, fibromyalgia, joint injuries, joint inflammation, joint degeneration, osteoporosis, organ transplant rejection, graft rejection, lupus,

uvetitis, Bechet's disease, Graves disease, Guillain-Barre syndrome, psoriasis, acute dermatomyositis, atopic skin disease, scleroderma, eczema, aplastic anemia, primary cirrhosis, autoimmune hepatitis, ulcerative colitis, Crohn's disease, amyotrophic lateral sclerosis, myasthenia gravis, multiple sclerosis, hepatic syndrome, glomerulonephritis, rheumatoid arthritis and diabetes mellitus; for reducing the risk of Sudden Infant Death Syndrome; for maintaining and effecting neuronal membrane ratios of phosphatidyl choline and cholesterol (all claimed). Also useful for treating e.g. septic shock, chronic fatigue syndrome, cachexia, head trauma, immune senescence, inflammatory bowel disorder, muscular dystrophy, neuropathic pain, nervous insult, peripheral nerve injury, renal failure, retinal ischemia, skin aging, diseases relating to lifespan and proliferative capacity of cells, diseases induced by cellular senescence, oxidative stress, age-related memory impairment, **ataxia-telangiectasia** syndrome, myocardial infarction, peripheral vasoconstriction, organ dysfunction, platelet consumption and activation, mitral valve pathology associated with acute perioperative pulmonary hypertension, chronic obstructive arterial disease, Raynaud's syndrome, renal artery stenosis, deep vein thrombosis, peripheral arterial occlusion, other blood stream thromboses, alloxan action, free fatty acid induced pancreatitis, abetalipoproteinemia, spontaneous abortion, infertility, sterility, sexual performance, post-menopausal syndrome, prostaglandin disorders, cataracts, ocular hemorrhage, degenerative retinal damage, retinopathy, endothelial injury, asthma, bronchitis, pneumonia, systemic lupus erythematosus, Zollinger-Ellison syndrome, gout, Batter's syndrome; and useful as dietary supplements.

ADVANTAGE - (I) Enhances activity of tocopherols, tocotrienols and the covalently linked compound in the relevant bio-chemical pathways that affects various conditions such as aging and longevity. (I) Decreases the release of superoxides by human peripheral blood neutrophils; reduces the levels of tumor necrosis factor and interleukin-1; increases antibody titers in blood; reduces total serum LDL-cholesterol, apolipoprotein B, thromboxane A2, platelet factor 4, triglycerides and glucose; and decreases lipoprotein A concentration in blood.

Dwg.0/0

FILE SEGMENT:	CPI
FIELD AVAILABILITY:	AB; GI; DCN
MANUAL CODES:	CPI: B03-A; B03-H; B04-B03A; B06-A01; B06-A03; B06-D09; B07-D09; B10-A04; B10-A17; B10-A22; B10-B02E; B10-B02J; B10-B03B; B10-C02; B10-C03; B10-C04C; B10-C04E; B10-E02; B14-C01; B14-C03; B14-C09; B14-D05D; B14-E08; B14-E10C; B14-E11; B14-E12; B14-F01; B14-F01B; B14-F01D; B14-F02; B14-F02B; B14-F02F3; B14-F03; B14-F04; B14-G01; B14-G02; B14-H01B; B14-J01A1; B14-J01A3; B14-J01A4; B14-J01B4; B14-J05; B14-K01; B14-K01A; B14-M01C; B14-N01; B14-N03; B14-N10; B14-N12; B14-N14; B14-N17; B14-P02; B14-S01; B14-S04; B14-S06; D08-B; E06-A01; E06-A03; E06-D09; E07-A02D; E07-D09A; E07-D12; E10-A04; E10-A17B; E10-A22G; E10-B02A1; E10-B02D6; E10-B03B1; E10-C02A; E10-C02F; E10-C03; E10-C04C; E10-C04L2; E10-E02E1; E10-E02F1; E10-E04M1
AN	2003-341309 [32] WPIX
DC	B02 D21 E13
IC	ICM A61K038-44
	ICS A61K031-714; A61K031-726
MC	CPI: B03-A; B03-H; B04-B03A; B06-A01; B06-A03; B06-D09; B07-D09; B10-A04; B10-A17; B10-A22; B10-B02E; B10-B02J; B10-B03B; B10-C02; B10-C03; B10-C04C; B10-C04E; B10-E02; B14-C01; B14-C03; B14-C09; B14-D05D; B14-E08; B14-E10C; B14-E11; B14-E12; B14-F01; B14-F01B; B14-F01D;

B14-F02; B14-F02B; B14-F02F3; B14-F03; B14-F04; B14-G01; B14-G02;
 B14-H01B; B14-J01A1; B14-J01A3; B14-J01A4; B14-J01B4; B14-J05;
 B14-K01; B14-K01A; B14-M01C; B14-N01; B14-N03; B14-N10; B14-N12;
 B14-N14; B14-N17; B14-P02; B14-S01; B14-S04; B14-S06; D08-B; E06-A01;
 E06-A03; E06-D09; E07-A02D; E07-D09A; E07-D12; E10-A04; E10-A17B;
 E10-A22G; E10-B02A1; E10-B02D6; E10-B03B1; E10-C02A; E10-C02F;
 E10-C03; E10-C04C; E10-C04L2; E10-E02E1; E10-E02F1; E10-E04M1

DRN 0048-S; 0048-U; 0053-S; 0053-U; 0058-S; 0058-U; 0091-S; 0091-U; 0118-S;
 0118-U; 0183-S; 0183-U; 0203-S; 0203-U; 0231-S; 0231-U; 0419-S; 0419-U;
 0829-S; 0829-U; 0902-S; 0902-U; 1090-S; 1090-U; 1170-S; 1170-U; 1210-S;
 1210-U; 1661-S; 1661-U

CMC UPB 20030522

M2 *01* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H402 H422 H8 J0 J011 J2
 J241 K0 L7 L730 L8 L812 L821 L831 L834 L943 M210 M211 M225
 M232 M240 M271 M281 M283 M311 M313 M321 M332 M342 M343 M349 M373
 M381 M391 M412 M512 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FC-T; RAA9FC-N

M2 *02* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H401 H421 H5 H521 H8 J0
 J011 J1 J171 K0 L7 L730 L8 L812 L821 L831 L834 L943 M1
 M125 M141 M210 M211 M225 M232 M240 M271 M281 M283 M311 M313 M321
 M332 M342 M343 M349 M373 M381 M391 M412 M512 M521 M530 M540 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FD-T; RAA9FD-N

M2 *03* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H401 H421 H5 H521 H8 J0
 J011 J1 J171 K0 L7 L730 L8 L812 L821 L831 L834 L943 M1
 M125 M141 M210 M211 M225 M232 M240 M271 M281 M283 M311 M313 M321
 M332 M342 M343 M349 M373 M381 M391 M412 M512 M521 M530 M540 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FE-T; RAA9FE-N

M2 *04* D012 D016 D022 D023 D024 D025 D120 H723 M210 M211 M225 M232 M240
 M281 M282 M283 M320 M412 M511 M520 M530 M540 M710 M904 M905 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: 0091-83101-T; 0091-83101-N

M2 *05* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H121 H2 H211
 H4 H403 H422 H481 H8 J5 J521 L9 L910 M210 M211 M240 M281
 M311 M321 M342 M373 M391 M413 M510 M522 M530 M540 M710 M904 M905
 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517
 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721
 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA76PC-T; RA76PC-Q; RA76PC-N

M2 *06* D011 D013 D931 F012 F013 F014 F015 F113 H1 H102 H122 H2 H201
 H4 H403 H422 H481 H5 H592 H8 H9 K0 L8 L812 L821 L834
 L910 M210 M211 M215 M232 M271 M273 M281 M311 M321 M342 M373 M391
 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423
 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
 P813 P816 P820 P822 P922 P943
 DCN: RAA9FG-T; RAA9FG-Q; RAA9FG-N

M2 *07* D011 D013 D931 F012 F013 F014 F015 F113 H1 H100 H122 H2 H201
 H4 H403 H422 H481 H8 L943 M210 M211 M240 M281 M311 M321 M342
 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA6YSO-T; RA6YSO-Q; RA6YSO-N

M2 *08* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H121 H2 H211
 H4 H402 H421 H481 H5 H521 H8 J5 J521 K0 L8 L812 L821
 L834 L9 L910 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413
 M510 M522 M530 M540 M710 M800 M904 M905 P220 P411 P420 P421 P423
 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
 P813 P816 P820 P822 P922 P943
 DCN: RA8CBT-T; RA8CBT-Q; RA8CBT-N

M2 *09* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H403 H422
 H481 H8 J5 J522 K0 L8 L812 L821 L834 L9 L910 M210 M211
 M240 M281 M311 M321 M342 M373 M391 M413 M510 M522 M530 M540 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R23688-T; R23688-Q; R23688-N

M2 *10* D011 D013 D931 F012 F013 F014 F015 F113 H1 H100 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 J5 J521 L8 L810 L811 L812
 L813 L821 L834 L9 L910 M210 M211 M272 M281 M311 M321 M342 M373
 M391 M412 M511 M521 M530 M540 M710 M800 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA8DA0-T; RA8DA0-Q; RA8DA0-N

M2 *11* D011 D019 D931 F012 F013 F014 F015 F113 H1 H100 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 L943 M210 M211 M272 M281 M311
 M321 M342 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA0TL1-T; RA0TL1-Q; RA0TL1-N

M2 *12* G015 G100 H4 H402 H441 H481 H5 H541 H8 J5 J581 M210 M211
 M272 M281 M312 M315 M321 M331 M332 M342 M372 M381 M391 M414 M510
 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: R20165-T; R20165-Q; R20165-N

M2 *13* G017 G100 H4 H401 H441 H8 M210 M211 M214 M233 M240 M283 M320
 M414 M510 M520 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R01090-T; R01090-Q; R01090-N; R16639-T; R16639-Q; R16639-N

M2 *14* H1 H181 J0 J012 J1 J171 J2 J271 K0 L7 L722 M210 M211
 M262 M273 M281 M283 M313 M321 M332 M343 M381 M391 M416 M620 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R07500-T; R07500-Q; R07500-N; R07501-T; R07501-Q; R07501-N

M2 *15* F012 F013 F014 F015 F019 F113 F542 H4 H402 H421 H481 H5 H521
 H8 J5 J522 K0 L8 L812 L821 L831 L835 L9 L910 M1 M116
 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522 M530
 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444

P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633
 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822
 P922 P943
 DCN: RAA9FL-T; RAA9FL-Q; RAA9FL-N

M2 *16* G015 G100 H4 H402 H441 H481 H5 H541 H8 J0 J011 J1 J171
 M210 M211 M272 M281 M311 M321 M343 M349 M371 M391 M414 M510 M520
 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: R00091-T; R00091-Q; R00091-N

M2 *17* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H402 H421
 H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
 L910 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522
 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434
 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625
 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820
 P822 P922 P943
 DCN: RAA9FM-T; RAA9FM-Q; RAA9FM-N

M2 *18* D011 D013 D931 F012 F013 F014 F015 F019 F113 H1 H103 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 J5 J521 K0 L8 L812 L821 L831 L834 L9
 L831 L834 L9 L910 M210 M211 M272 M281 M282 M311 M321 M342
 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FV-T; RAA9FV-Q; RAA9FV-N

M2 *19* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H402 H421
 H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
 L910 M210 M211 M240 M272 M281 M311 M321 M342 M373 M391 M413 M510
 M522 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: RAA9FW-T; RAA9FW-Q; RAA9FW-N

M2 *20* D011 D019 D931 F012 F013 F014 F015 F019 F113 H1 H102 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 K0 L8 L812 L821 L831 L834
 L943 M210 M211 M272 M273 M281 M311 M321 M342 M373 M391 M412 M511
 M521 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: RAA9FX-T; RAA9FX-Q; RAA9FX-N

M2 *21* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H181 H2 H212
 H4 H403 H422 H481 H8 J0 J011 J1 J171 J5 J522 K0 L8
 L812 L821 L834 L9 L910 M280 M311 M313 M321 M332 M342 M343 M349
 M373 M381 M391 M413 M510 M522 M530 M540 M710 M904 M905 P220 P411
 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FY-T; RAA9FY-Q; RAA9FY-N

M2 *22* H1 H181 J0 J011 J2 J271 K0 L7 L722 M210 M211 M262 M273
 M281 M283 M312 M321 M332 M342 M383 M391 M416 M620 M710 M904 M905
 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714
 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00058-T; R00058-Q; R00058-N

M2 *23* C216 H7 H716 H723 K0 K2 K224 K4 K442 M210 M213 M231 M271
 M282 M313 M321 M332 M342 M383 M391 M416 M710 M904 M905 P220 P411
 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520

P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R06395-T; R06395-Q; R06395-N

M2 *24* H1 H100 H181 J0 J011 J1 J171 M280 M312 M321 M331 M340 M342
 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R01210-T; R01210-Q; R01210-N; R10414-T; R10414-Q; R10414-N

M2 *25* G030 G038 G530 H1 H100 H161 J0 J011 J1 J151 M280 M320 M415
 M510 M520 M530 M541 M710 M904 M905 P220 P411 P420 P421 P423 P431
 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813
 P816 P820 P822 P922 P943
 DCN: R06983-T; R06983-Q; R06983-N

M2 *26* F011 F015 F521 H1 H100 H182 H2 H201 J0 J012 J1 J171 J3
 J371 M210 M211 M273 M281 M312 M322 M332 M342 M343 M349 M371 M381
 M391 M413 M510 M521 M530 M540 M710 M800 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R11742-T; R11742-Q; R11742-N

M2 *27* D013 D023 D120 G013 G100 H4 H403 H443 H8 J5 J521 M1 M113
 M280 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R06069-T; R06069-Q; R06069-N

M2 *28* H7 H723 J0 J011 J1 J171 M226 M231 M262 M281 M320 M416 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P822 P922 P943
 DCN: R04038-T; R04038-Q; R04038-N; R13711-T; R13711-Q; R13711-N

M2 *29* H1 H100 H181 J0 J011 J1 J171 K0 L2 L250 M280 M314 M321
 M332 M343 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411
 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R01661-T; R01661-Q; R01661-N; R04740-T; R04740-Q; R04740-N

M2 *30* G037 G038 G039 G562 G599 H4 H402 H462 H7 H725 H8 J5 J562
 M1 M126 M135 M210 M211 M240 M283 M316 M321 M333 M342 M415 M510
 M520 M530 M542 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: R11112-T; R11112-Q; R11112-N

M2 *31* D012 D013 D940 H1 H100 H121 H4 H402 H482 H8 J5 J521 L9
 L910 M280 M313 M321 M331 M343 M373 M391 M412 M511 M520 M530 M540
 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
 P943
 DCN: RA1717-T; RA1717-Q; RA1717-N

M2 *32* H1 H181 J0 J011 J1 J171 K0 L7 L722 M210 M211 M273 M283
 M311 M321 M342 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00829-T; R00829-Q; R00829-N

M2 *33* H1 H181 H4 H401 H481 H8 J0 J011 J1 J171 K0 L7 L722

M210 M211 M273 M283 M313 M321 M332 M343 M381 M391 M416 M620 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R12266-T; R12266-Q; R12266-N

M2 *34* F014 F521 H1 H100 H181 J0 J012 J1 J171 J3 J371 M280 M312
 M322 M332 M342 M343 M349 M371 M381 M391 M413 M510 M521 M530 M540
 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
 P943
 DCN: R08807-T; R08807-Q; R08807-N

M2 *35* D013 D023 D120 G015 G100 H4 H405 H421 H444 H8 M1 M113 M280
 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R04686-T; R04686-Q; R04686-N

M2 *36* G015 G037 G038 G111 G563 H4 H405 H442 H463 H7 H721 H8 J0
 J012 J1 J151 J2 J261 K0 L8 L818 L821 L832 M280 M312 M321
 M332 M342 M372 M391 M414 M510 M520 M531 M541 M710 M904 M905 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R12870-T; R12870-Q; R12870-N

M2 *37* H1 H181 H4 H401 H481 H8 K0 L7 L722 M210 M211 M273 M283
 M312 M321 M332 M342 M383 M391 M416 M620 M710 M904 M905 M910 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00231-T; R00231-Q; R00231-N; R04247-T; R04247-Q; R04247-N

M2 *38* H4 H401 H481 H8 J0 J013 J1 J173 M280 M313 M321 M332 M344
 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R00419-T; R00419-Q; R00419-N; R07029-T; R07029-Q; R07029-N

M2 *39* J0 J011 J1 J171 K0 L2 L250 M210 M211 M273 M281 M311 M321
 M342 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00118-T; R00118-Q; R00118-N

M2 *40* F011 F012 F014 F522 H1 H100 H121 H181 H2 H201 J5 J521 L9
 L910 M210 M211 M273 M281 M320 M413 M510 M521 M530 M540 M710 M904
 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00203-T; R00203-Q; R00203-N

M2 *41* G013 G100 J0 J011 J1 J131 M210 M213 M232 M240 M281 M320 M414
 M510 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431
 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813
 P816 P820 P822 P922 P943
 DCN: R16027-T; R16027-Q; R16027-N

M2 *42* D013 D022 D120 G013 G100 H4 H402 H442 H8 J5 J521 M1 M113
 M280 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943

M2 *43* DCN: RA00TD-T; RA00TD-Q; RA00TD-N
 G015 G100 H1 H100 H181 H4 H402 H442 H8 M280 M312 M321 M332
 M342 M373 M391 M414 M510 M520 M531 M540 M710 M904 M905 M910 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00053-T; R00053-Q; R00053-N

M2 *44* D011 D019 D023 D029 D240 H4 H404 H444 H8 J5 J522 L9 L942
 L999 M280 M320 M412 M511 M520 M530 M540 M710 M904 M905 P220 P411
 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
 P738 P811 P812 P813 P816 P820 P822 P922 P943
 RIN: 05197 05197
 DCN: R17082-T; R17082-Q; R17082-N; R17083-T; R17083-Q; R17083-N

M2 *45* D013 D023 D120 G017 G100 H4 H405 H421 H444 H8 M1 M113 M280
 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P820 P822 P922 P943
 DCN: RA00TN-T; RA00TN-Q; RA00TN-N

M2 *46* G015 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M211 M273
 M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540 M710
 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
 P943
 DCN: R00048-T; R00048-Q; R00048-N; R14840-T; R14840-Q; R14840-N

M2 *47* D012 D940 G013 G100 H1 H100 H102 H121 H141 J0 J013 J1
 J172 J3 J331 J5 J521 L9 L910 M280 M311 M313 M321 M332 M342
 M343 M349 M373 M381 M391 M412 M511 M520 M531 M540 M710 M904 M905
 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714
 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00183-T; R00183-Q; R00183-N

M2 *48* H7 H721 J0 J012 J1 J172 M280 M312 M321 M332 M342 M382 M391
 M416 M710 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434
 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625
 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820
 P822 P922 P943
 DCN: R00902-T; R00902-Q; R00902-N; R04891-T; R04891-Q; R04891-N

M2 *49* G017 G100 H4 H403 H443 H8 J0 J011 J1 J131 M280 M320 M414
 M510 M520 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421 P423
 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
 P813 P816 P820 P822 P922 P943
 DCN: R01170-T; R01170-Q; R01170-N; R09472-T; R09472-Q; R09472-N

M3 *01* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H402 H422 H8 J0 J011 J2
 J241 K0 L7 L730 L8 L812 L821 L831 L834 L943 M210 M211 M225
 M232 M240 M271 M281 M283 M311 M313 M321 M332 M342 M343 M349 M373
 M381 M391 M412 M512 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FC-T; RAA9FC-N

M3 *02* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H401 H421 H5 H521 H8 J0
 J011 J1 J171 K0 L7 L730 L8 L812 L821 L831 L834 L943 M1
 M125 M141 M210 M211 M225 M232 M240 M271 M281 M283 M311 M313 M321
 M332 M342 M343 M349 M373 M381 M391 M412 M512 M521 M530 M710

M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FD-T; RAA9FD-N

M3 *03* C216 D011 D012 D016 D019 D025 D120 D931 F012 F013 F014 F015 F113
 H1 H101 H122 H181 H2 H201 H4 H401 H421 H5 H521 H8 J0
 J011 J1 J171 K0 L7 L730 L8 L812 L821 L831 L834 L943 M1
 M125 M141 M210 M211 M225 M232 M240 M271 M281 M283 M311 M313 M321
 M332 M342 M343 M349 M373 M381 M391 M412 M512 M521 M530 M540 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RAA9FE-T; RAA9FE-N

M3 *04* D012 D016 D022 D023 D024 D025 D120 H723 M210 M211 M225 M232 M240
 M281 M282 M283 M320 M412 M511 M520 M530 M540 M710 M904 M905 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: 0091-83101-T; 0091-83101-N

M3 *05* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H121 H2 H211
 H4 H403 H422 H481 H8 J5 J521 L9 L910 M210 M211 M240 M281
 M311 M321 M342 M373 M391 M413 M510 M522 M530 M540 M710 M904 M905
 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517
 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721
 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA76PC-T; RA76PC-Q; RA76PC-N

M3 *06* D011 D013 D931 F012 F013 F014 F015 F019 F113 H1 H102 H122 H2 H201
 H4 H403 H422 H481 H5 H592 H8 H9 K0 L8 L812 L821 L834
 L910 M210 M211 M215 M232 M271 M273 M281 M311 M321 M342 M373 M391
 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423
 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
 P813 P816 P820 P822 P922 P943
 DCN: RAA9FG-T; RAA9FG-Q; RAA9FG-N

M3 *07* D011 D013 D931 F012 F013 F014 F015 F019 F113 H1 H100 H122 H2 H201
 H4 H403 H422 H481 H8 L943 M210 M211 M240 M281 M311 M321 M342
 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA6YSO-T; RA6YSO-Q; RA6YSO-N

M3 *08* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H121 H2 H211
 H4 H402 H421 H481 H5 H521 H8 J5 J521 K0 L8 L812 L821 L834
 L834 L9 L910 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413
 M510 M522 M530 M540 M710 M800 M904 M905 P220 P411 P420 P421 P423
 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
 P813 P816 P820 P822 P922 P943
 DCN: RA8CBT-T; RA8CBT-Q; RA8CBT-N

M3 *09* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H403 H422
 H481 H8 J5 J522 K0 L8 L812 L821 L834 L9 L910 M210 M211
 M240 M281 M311 M321 M342 M373 M391 M413 M510 M522 M530 M540 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R23688-T; R23688-Q; R23688-N

M3 *10* D011 D013 D931 F012 F013 F014 F015 F019 F113 H1 H100 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 J5 J521 L8 L810 L811 L812
 L813 L821 L834 L9 L910 M210 M211 M272 M281 M311 M321 M342 M373
 M391 M412 M511 M521 M530 M540 M710 M800 M904 M905 P220 P411 P420

P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA8DA0-T; RA8DA0-Q; RA8DA0-N

M3 *11* D011 D019 D931 F012 F013 F014 F015 F113 H1 H100 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 L943 M210 M211 M272 M281 M311
 M321 M342 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA0TL1-T; RA0TL1-Q; RA0TL1-N

M3 *12* G015 G100 H4 H402 H441 H481 H5 H541 H8 J5 J581 M210 M211
 M272 M281 M312 M315 M321 M331 M332 M342 M372 M381 M391 M414 M510
 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
 P434 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: R20165-T; R20165-Q; R20165-N

M3 *13* G017 G100 H4 H401 H441 H8 M210 M211 M214 M233 M240 M283 M320
 M414 M510 M520 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421
 P423 P431 P433 P434 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R01090-T; R01090-Q; R01090-N; R16639-T; R16639-Q; R16639-N

M3 *14* H1 H181 J0 J012 J1 J171 J2 J271 K0 L7 L722 M210 M211
 M262 M273 M281 M283 M313 M321 M332 M343 M381 M391 M416 M620 M710
 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R07500-T; R07500-Q; R07500-N; R07501-T; R07501-Q; R07501-N

M3 *15* F012 F013 F014 F015 F019 F113 F542 H4 H402 H421 H481 H5 H521
 H8 J5 J522 K0 L8 L812 L821 L831 L835 L9 L910 M1 M116
 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522 M530
 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444
 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633
 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822
 P922 P943
 DCN: RAA9FL-T; RAA9FL-Q; RAA9FL-N

M3 *16* G015 G100 H4 H402 H441 H481 H5 H541 H8 J0 J011 J1 J171
 M210 M211 M272 M281 M311 M321 M343 M349 M371 M391 M414 M510 M520
 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433
 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
 P820 P822 P922 P943
 DCN: R00091-T; R00091-Q; R00091-N

M3 *17* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H402 H421
 H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
 L910 M210 M211 M272 M281 M311 M321 M342 M373 M391 M413 M510 M522
 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434
 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625
 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820
 P822 P922 P943
 DCN: RAA9FM-T; RAA9FM-Q; RAA9FM-N

M3 *18* D011 D013 D931 F012 F013 F014 F015 F113 H1 H103 H122 H2 H201
 H4 H402 H421 H481 H5 H521 H8 J5 J521 K0 L8 L812 L821
 L831 L834 L9 L910 M210 M211 M272 M281 M282 M311 M321 M342
 M373 M391 M412 M511 M521 M530 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943

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DCN: RAA9FV-T; RAA9FV-Q; RAA9FV-N
M3 *19* F011 F012 F013 F014 F015 F019 F113 F542 H2 H211 H4 H402 H421
H481 H5 H521 H8 J5 J522 K0 L8 L812 L821 L831 L834 L9
L910 M210 M211 M240 M272 M281 M311 M321 M342 M373 M391 M413 M510
M522 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
P820 P822 P922 P943
DCN: RAA9FW-T; RAA9FW-Q; RAA9FW-N
M3 *20* D011 D019 D931 F012 F013 F014 F015 F113 H1 H102 H122 H2 H201
H4 H402 H421 H481 H5 H521 H8 K0 L8 L812 L821 L831 L834
L943 M210 M211 M272 M273 M281 M311 M321 M342 M373 M391 M412 M511
M521 M530 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
P820 P822 P922 P943
DCN: RAA9FX-T; RAA9FX-Q; RAA9FX-N
M3 *21* F011 F012 F013 F014 F015 F019 F113 F542 H1 H100 H181 H2 H212
H4 H403 H422 H481 H8 J0 J011 J1 J171 J5 J522 K0 L8
L812 L821 L834 L9 L910 M280 M311 M313 M321 M332 M342 M343 M349
M373 M381 M391 M413 M510 M522 M530 M540 M710 M904 M905 P220 P411
P420 P421 P423 P431 P433 P434 P446 P448 P451 P517 P519 P520
P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: RAA9FY-T; RAA9FY-Q; RAA9FY-N
M3 *22* H1 H181 J0 J011 J2 J271 K0 L7 L722 M210 M211 M262 M273
M281 M283 M312 M321 M332 M342 M383 M391 M416 M620 M710 M904 M905
M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714
P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R00058-T; R00058-Q; R00058-N
M3 *23* C216 H7 H716 H723 K0 K2 K224 K4 K442 M210 M213 M231 M271
M282 M313 M321 M332 M342 M383 M391 M416 M710 M904 M905 P220 P411
P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R06395-T; R06395-Q; R06395-N
M3 *24* H1 H100 H181 J0 J011 J1 J171 M280 M312 M321 M331 M340 M342
M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
P812 P813 P816 P820 P822 P922 P943
DCN: R01210-T; R01210-Q; R01210-N; R10414-T; R10414-Q; R10414-N
M3 *25* G030 G038 G530 H1 H100 H161 J0 J011 J1 J151 M280 M320 M415
M510 M520 M530 M541 M710 M904 M905 P220 P411 P420 P421 P423 P431
P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813
P816 P820 P822 P922 P943
DCN: R06983-T; R06983-Q; R06983-N
M3 *26* F011 F015 F521 H1 H100 H182 H2 H201 J0 J012 J1 J171 J3
J371 M210 M211 M273 M281 M312 M322 M332 M342 M343 M349 M371 M381
M391 M413 M510 M521 M530 M540 M710 M800 M904 M905 P220 P411 P420
P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
P811 P812 P813 P816 P820 P822 P922 P943
DCN: R11742-T; R11742-Q; R11742-N
M3 *27* D013 D023 D120 G013 G100 H4 H403 H443 H8 J5 J521 M1 M113
M280 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420
P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738

P811 P812 P813 P816 P820 P822 P922 P943
DCN: R06069-T; R06069-Q; R06069-N

M3 *28* H7 H723 J0 J011 J1 J171 M226 M231 M262 M281 M320 M416 M710
M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
P714 P721 P723 P731 P738 P811 P812 P813 P816 P818 P820 P822 P922 P943
DCN: R04038-T; R04038-Q; R04038-N; R13711-T; R13711-Q; R13711-N

M3 *29* H1 H100 H181 J0 J011 J1 J171 K0 L2 L250 M280 M314 M321
M332 M343 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411
P420 P421 P423 P431 P434 P444 P446 P448 P451 P517 P519 P520
P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R01661-T; R01661-Q; R01661-N; R04740-T; R04740-Q; R04740-N

M3 *30* G037 G038 G039 G562 G599 H4 H402 H462 H7 H725 H8 J5 J562
M1 M126 M135 M210 M211 M240 M283 M316 M321 M333 M342 M415 M510
M520 M530 M542 M710 M904 M905 P220 P411 P420 P421 P423 P431 P433
P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616
P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816
P820 P822 P922 P943
DCN: R11112-T; R11112-Q; R11112-N

M3 *31* D012 D013 D940 H1 H100 H121 H4 H402 H482 H8 J5 J521 L9
L910 M280 M313 M321 M331 M343 M373 M391 M412 M511 M520 M530 M540
M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
P943
DCN: RA1717-T; RA1717-Q; RA1717-N

M3 *32* H1 H181 J0 J011 J1 J171 K0 L7 L722 M210 M211 M273 M283
M311 M321 M342 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220
P411 P420 P421 P423 P431 P434 P444 P446 P448 P451 P517 P519
P520 P522 P523 P526 P616 P625 P633 P646 P711 P714 P721 P723
P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R00829-T; R00829-Q; R00829-N

M3 *33* H1 H181 H4 H401 H481 H8 J0 J011 J1 J171 K0 L7 L722
M210 M211 M273 M283 M313 M321 M332 M343 M381 M391 M416 M620 M710
M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R12266-T; R12266-Q; R12266-N

M3 *34* F014 F521 H1 H100 H181 J0 J012 J1 J171 J3 J371 M280 M312
M322 M332 M342 M349 M371 M381 M391 M413 M510 M521 M530 M540
M710 M904 M905 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
P943
DCN: R08807-T; R08807-Q; R08807-N

M3 *35* D013 D023 D120 G015 G100 H4 H405 H421 H444 H8 M1 M113 M280
M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
P812 P813 P816 P820 P822 P922 P943
DCN: R04686-T; R04686-Q; R04686-N

M3 *36* G015 G037 G038 G111 G563 H4 H405 H442 H463 H7 H721 H8 J0
J012 J1 J151 J2 J261 K0 L8 L818 L821 L832 M280 M312 M321
M332 M342 M372 M391 M414 M510 M520 M531 M541 M710 M904 M905 P220
P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R12870-T; R12870-Q; R12870-N

M3 *37* H1 H181 H4 H401 H481 H8 K0 L7 L722 M210 M211 M273 M283

M312 M321 M332 M342 M383 M391 M416 M620 M710 M904 M905 M910 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519
 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00231-T; R00231-Q; R00231-N; R04247-T; R04247-Q; R04247-N

M3 *38* H4 H401 H481 H8 J0 J013 J1 J173 M280 M313 M321 M332 M344
 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: R00419-T; R00419-Q; R00419-N; R07029-T; R07029-Q; R07029-N

M3 *39* J0 J011 J1 J171 K0 L2 L250 M210 M211 M273 M281 M311 M321
 M342 M349 M381 M391 M416 M620 M710 M904 M905 M910 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00118-T; R00118-Q; R00118-N

M3 *40* F011 F012 F014 F522 H1 H100 H121 H181 H2 H201 J5 J521 L9
 L910 M210 M211 M273 M281 M320 M413 M510 M521 M530 M540 M710 M904
 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448
 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711
 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00203-T; R00203-Q; R00203-N

M3 *41* G013 G100 J0 J011 J1 J131 M210 M213 M232 M240 M281 M320 M414
 M510 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421 P423 P431
 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528
 P616 P625 P633 P646 P711 P721 P723 P731 P738 P811 P812 P813
 P816 P820 P822 P922 P943
 DCN: R16027-T; R16027-Q; R16027-N

M3 *42* D013 D022 D120 G013 G100 H4 H402 H442 H8 J5 J521 M1 M113
 M280 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420
 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: RA00TD-T; RA00TD-Q; RA00TD-N

M3 *43* G015 G100 H1 H100 H181 H4 H402 H442 H8 M280 M312 M321 M332
 M342 M373 M391 M414 M510 M520 M531 M540 M710 M904 M905 M910 P220
 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738
 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
 DCN: R00053-T; R00053-Q; R00053-N

M3 *44* D011 D019 D023 D029 D240 H4 H404 H444 H8 J5 J522 L9 L942
 L999 M280 M320 M412 M511 M520 M530 M540 M710 M904 M905 P220 P411
 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520
 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731
 P738 P811 P812 P813 P816 P820 P822 P922 P943
 RIN: 05197 05197
 DCN: R17082-T; R17082-Q; R17082-N; R17083-T; R17083-Q; R17083-N

M3 *45* D013 D023 D120 G017 G100 H4 H405 H421 H444 H8 M1 M113 M280
 M320 M412 M511 M520 M531 M540 M710 M904 M905 P220 P411 P420 P421
 P423 P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523
 P526 P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811
 P812 P813 P816 P820 P822 P922 P943
 DCN: RA00TN-T; RA00TN-Q; RA00TN-N

M3 *46* G015 G100 H1 H102 H181 H4 H403 H442 H481 H8 M210 M211 M273
 M281 M312 M321 M332 M343 M373 M391 M414 M510 M520 M531 M540 M710
 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446
 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646
 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922
 P943

M3 *47* DCN: R00048-T; R00048-Q; R00048-N; R14840-T; R14840-Q; R14840-N
D012 D013 D940 G013 G100 H1 H100 H102 H121 H141 J0 J013 J1
J172 J3 J331 J5 J521 L9 L910 M280 M311 M313 M321 M332 M342
M343 M349 M373 M381 M391 M412 M511 M520 M531 M540 M710 M904 M905
M910 P220 P411 P420 P421 P423 P431 P433 P434 P444 P446 P448 P451
P517 P519 P520 P522 P523 P526 P528 P616 P625 P633 P646 P711 P714
P721 P723 P731 P738 P811 P812 P813 P816 P820 P822 P922 P943
DCN: R00183-T; R00183-Q; R00183-N

M3 *48* H7 H721 J0 J012 J1 J172 M280 M312 M321 M332 M342 M382 M391
M416 M710 M904 M905 M910 P220 P411 P420 P421 P423 P431 P433 P434
P444 P446 P448 P451 P517 P519 P520 P522 P523 P526 P528 P616 P625
P633 P646 P711 P714 P721 P723 P731 P738 P811 P812 P813 P816 P820
P822 P922 P943
DCN: R00902-T; R00902-Q; R00902-N; R04891-T; R04891-Q; R04891-N

M3 *49* G017 G100 H4 H403 H443 H8 J0 J011 J1 J131 M280 M320 M414
M510 M520 M531 M540 M710 M904 M905 M910 P220 P411 P420 P421 P423
P431 P433 P434 P444 P446 P448 P451 P517 P519 P520 P522 P523 P526
P528 P616 P625 P633 P646 P711 P714 P721 P723 P731 P738 P811 P812
P813 P816 P820 P822 P922 P943
DCN: R01170-T; R01170-Q; R01170-N; R09472-T; R09472-Q; R09472-N

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